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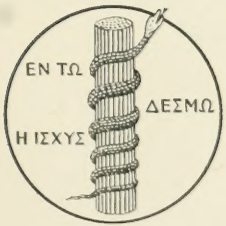

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EDINBURGH MEDICAL JOURNAL.

EDITORIAL NOTES.

Educative Convalescence for the Wounded.

UNDER this suggestive title has been published a small brochure descriptive of a scheme which has been carried out at the Heritage Craft Schools, Chailey, Sussex. In times of peace this institution is a school for cripple children in which the underlying idea in education is to furnish the cripples with a means of earning a livelihood in such a way as to make life for them worth living. Under skilled teachers each child receives a training in some art or craft adapted to his particular disability and within his mental and physical capacity.

"Last summer saw a novel experiment at the Home towards repairing the wastage of war. A batch of sorely crippled soldiers in the earliest stages of convalescence was brought to Chailey. To each was assigned as orderlies two of the trained crippled boys suffering from the same loss of limb. The boys became the soldier's nurses and instructors. He learnt from them how to get on as well as they did without a right arm or whatever it might be. When convalescence was established the soldiers were led by curiosity to follow their orderlies to the workshop and watch them at work. Not at all keen to do anything but laze, they found the nimbleness and the skill of the carpenters and metal-workers and basket-makers, and what not, very catching. Before they quite knew what was happening, they had caught the way of the thing, and to their own surprise they were useful workmen."

The problem of providing for the multitudes of maimed which the war will leave with us is one of the greatest that will have to be faced and overcome. Alike on grounds of national economy and for the happiness of the victims, the maimed and crippled must be rendered useful and productive members of the community. Their material requirements must be secured to them by the State without stint or grudge. Their moral welfare will best be ensured by providing them with a means of employing their time and capacities usefully, and

of taking their share in restoring, as they have already taken their share in preserving, the fabric of the State. Steps are already being taken in military convalescent camps to initiate this process, and the results of the work being carried out under the auspices of the War Office will be watched with great interest.

The experiment, carried out on a comparatively small scale at Chailey, raises high hopes. Already a number of soldiers sorely handicapped by the loss of a limb or in other ways have acquired sufficient dexterity in such crafts as bookbinding, basket-making, rugmaking, copper work, leather work, or toymaking, as to render them almost self-supporting. The carrying out of this useful work has entailed heavy expense on the part of the Chailey schools, but the results have been so satisfactory that they are anxious to continue and extend it, and for this purpose they appeal for funds.

CASUALTIES.

KILLED in action, Lieutenant-Colonel GEORGE OLIVER MOORHEAD, South African Imperial Force, Nyasaland Contingent.

Lieutenant-Colonel Moorhead was educated at Malines in Belgium and at Edinburgh University, where he graduated in medicine. He subsequently went out to South Africa, and was practising in the Transvaal.

KILLED in action, Lieutenant JAMES M'C. JOHNSTON, R.A.M.C., aged 32.

Lieutenant Johnston studied at the Universities of Edinburgh and St. Andrews, and graduated M.B., Ch.B. at the latter University in 1913.

KILLED in action on 31st May 1916, Surgeon CHARLES W. LEWIS, H.M.S. *Queen Mary*.

Surgeon Lewis graduated M.B., Ch.B.(Edin.) in 1914, and received his commission as temporary Surgeon, R.N., on 13th March 1915.

KILLED in action on 31st May 1916, Surgeon A. A. MORISON, H.M.S. *Indefatigable*, aged 29.

Surgeon Morison was educated at the University of Edinburgh, where he graduated M.B., Ch.B. in 1912. He received a temporary commission as Surgeon, R.N., on 6th August 1914.

KILLED in action on 31st May 1916, Fleet-Surgeon FREDERICK A. CAPPS, H.M.S. *Defence*.

Fleet-Surgeon Capps received his medical education in Edinburgh, and took the Triple Scottish Qualification in 1890. He attained the rank of Fleet-Surgeon on 11th May 1908.

KILLED in action on 31st May 1916, Surgeon JOHN S. D. MACCORMAC, H.M.S. *Black Prince*, aged 44.

Surgeon MacCormac took the Scottish Triple Qualification in 1894. On the outbreak of war he received a commission as Surgeon.

KILLED in action, Surgeon GEORGE B. MOON, R.N.

Surgeon Moon was educated at Birmingham and Edinburgh, and took the Triple Scottish Qualification in 1909. He joined the Navy with a temporary commission on 27th November 1914.

DIED of wounds on 29th May, Captain WILLIAM NORMAN WATSON, K.O.S.B., aged 27.

Captain Watson received his education at Edinburgh University, where he graduated M.B., Ch.B. in 1913. He joined the R.A.M.C. in December 1914. Captain Watson won the Military Cross at the battle of Loos, and at the request of the commanding officer he took a combatant commission in April last.

DIED on service, Temporary Captain ELFRED CHALMERS AUSTIN, R.A.M.C., on 5th June, aged 39.

Captain Austin was educated at St. Mary's Hospital, and took the diplomas of M.R.C.S. and L.R.C.P.(Lond.) in 1899, subsequently taking the F.R.C.S. at Edinburgh in 1903.

MEDICAL STUDENTS.

KILLED in action on 31st May 1916, Surgeon-Probationer ROBERT WALKER, H.M.S. *Shark*.

Surgeon-Probationer Walker was in his fourth year as a student in medicine at the University of Edinburgh. He had won the Vans Dunlop scholarship in anatomy, the John Aitken Carlyle and the Mackenzie bursaries in anatomy as well as the Cunningham memorial medal presented to the most brilliant student of the year in anatomy. He had in addition won eight medals in other subjects at the University.

DIED of wounds in France, Second Lieutenant ANDREW J. AITCHISON, Highland Light Infantry, aged about 27.

Lieutenant Aitchison was studying medicine at the Glasgow University when he joined the colours over a year ago.

WAR HONOURS.

THE following honours have been conferred on members of the medical services.

The Victoria Cross has been awarded to Captain JOHN ALEXANDER SINTON, M.B., I.M.S., for conspicuous bravery and devotion to duty.

Although shot through both arms and through the side he refused to go the hospital, and remained, as long as daylight lasted, attending to his duties under very heavy fire. In three previous actions Captain Sinton displayed the utmost bravery. Captain Sinton was a student at Queen's College, Belfast.

C.M.G.—Lieutenant-Colonel J. V. Forrest, M.B., R.A.M.C.: Lieutenant-Colonel G. S. Thom, M.B., R.A.M.C.

Distinguished Service Order.—Major John May Darling, M.B., F.R.C.S., R.A.M.C. (S.R.): Major A. N. Fraser, M.B., R.A.M.C.: Major P. H. Henderson, M.B., R.A.M.C.; Captain G. S. Husband, M.B., I.M.S.

Military Cross.—Captain Gurth S. Blandy, M.D., R.A.M.C.; Captain J. C. Brash, M.A., B.Sc., M.B., R.A.M.C.: Captain Alex. Bremner, M.B., R.A.M.C.; Captain L. C. Bruce, M.D., R.A.M.C. (T.); Captain G. R. Grant, M.B., R.A.M.C.: Captain Arthur C. Keep, M.D., R.A.M.C.: Captain H. F. Panton, M.B., R.A.M.C.; Captain M. A. Swan, M.B., R.A.M.C.

To be Brevet Lieutenant-Colonel.—Major (Temporary Brigadier-General) A. C. Geddes, unattached list (T.F.), M.D., Director of Recruiting.

SYPHILITIC DISEASES OF THE EAR.

A Résumé of Recent Literature.

By J. S. FRASER, F.R.C.S.,
Assistant Surgeon, Ear Department, Royal Infirmary, Edinburgh.

I. Introduction.

Owing to the difficulty of obtaining pathological material, and of carrying out the microscopic examination of the auditory apparatus, our knowledge of the pathology of syphilitic diseases of the ear is still very meagre. The importance of the subject is undoubtedly great. Congenital syphilis ranks after epidemic cerebro-spinal meningitis and middle-ear suppuration as the most frequent cause of acquired deafmutism. There is reason also to believe that many cases of "congenital" deafmutism are really due to intra-uterine syphilis or to syphilitic changes in the ear, occurring before the child has learned to talk. Statistics as to the frequency of deafness in children suffering from congenital syphilis vary very greatly. Some authorities—Carpenter—put it as high as 60 per cent., others at only 33 per cent.

Yearsley found that 3·5 per cent. of 500 children in the L. C. C. deafmute schools were cases of congenital syphilis, Siebenmann of Basle found that 5·6 per cent. of cases of inherited deafmutism were due to syphilis, while Bock (*Munch. med. Wochenschr.*, 1910, p. 2083) obtained a percentage of 8·1. E. Urbantschitsch has examined the Wassermann reaction in 125 deafmutes and found it positive in 3%.

Acquired syphilitic disease of the ear is also of importance, on account of the frequent occurrence of sudden and severe deafness in the secondary and tertiary stages. Further, it has recently been found that one third of the cases of nerve-deafness of unexplained origin have a positive Wassermann reaction—thus pointing to a probably syphilitic origin.

II. Congenital Syphilis.

A. *Pathology.*

1. *In the Fœtus and in Infants.*—Baratoux, Panse, Mayer, Asai, Hofer, Ranke, and Gruenberg have microscopically examined the middle and inner ears of syphilitic fœtuses and infants. The changes found by these observers may be summarised as follows:—(a) *Otitis media* is of common occurrence not only in infants but also in children born prematurely. In some cases the infective processes *involve the labyrinth* by rupture of the annular ligament and consequent invasion of the

vestibule through the oval window. (*B*) *Delayed ossification* of the petrous bone, with *abnormal narrow spaces*. (*c*) Intra-uterine *meningitis* and *neuritis* of the eighth nerve. (*d*) Baratoux alone has noted changes in the vessels, though he also found purulent otitis media and interna. (*e*) Hæmorrhages in the middle and inner ear have often been observed, but such findings must be discounted, as they were probably due to suffocation. (*f*) Panse, however, holds that the hæmorrhage is of syphilitic origin. Changes in Corti's organ and in other parts of the membranous labyrinth and nerve apparatus, especially in the spiral ganglion, have occasionally been noted, but may in some cases be explained by post-mortem changes. Gruenberg has demonstrated spirochætes in microscopic sections from the ear of a seven-months' fœtus, especially in the cochlear and vestibular nerves, the facial nerve, and in the tympanic and carotid plexuses. Spirochætes were also found in the neighbourhood of the vessels of the middle ear, as well as in the narrow spaces of the ossicles. There were no spirochætes in the labyrinth or in the nerve endings in the inner ear. Alexander states that the most severe forms of ear syphilis occur in intra-uterine life. The new-born infants show all the signs of congenital deafness, and the static labyrinth is not excitable. The abstractor has examined both ears from a syphilitic fœtus of seven months, and found otitis media on both sides; abnormal narrow spaces between the cartilage and periosteal bone; small cell infiltration of the eighth nerve. Changes were present in the membranous labyrinth, but these appeared to be of a "post-mortem" nature.

2. *In Young Children.*—M'Bride (*Diseases of the Throat, Nose, and Ear*) states that, in children suffering from congenital syphilis, what appears to be a case of simple Eustachian catarrh, runs an unfavourable course. Treatment does no good, and the drumheads remain thickened and indrawn while the deafness is severe and persists. M'Bride further says that in children hereditary syphilis often gives rise to a combination of middle ear catarrh and labyrinthine deafness. From this it would appear that, if cases of middle ear catarrh do not rapidly clear up—especially if they are accompanied by severe deafness—it is advisable to have the Wassermann or Noguchi reaction tested.

Alexander (*Oberndorfchandb des Kinderalters*) says that middle ear catarrh is common in syphilitic children and appears to be a syphilitic manifestation. There is only slight or no improvement after the absorption of the exudate. The middle ear symptoms are thus replaced by those of an inner-ear lesion. These catarrhal affections develop into conditions which clinically correspond to otosclerosis. The prognosis is not good on account of the poor nourishment of the child. According to Cheatle, Eustachian obstruction occurs in the "snuffling" stage of congenital syphilis. Cheatle holds that syphilis alone will not cause suppurative otitis media: pyogenic organisms are necessary.

Urban Pritchard calls attention to those cases of deafness in children where removal of adenoids does no good. He holds that these are due to syphilis. Madden (quoted by Cheatle) describes a case of gumma of the middle ear in which there was a deep opening behind the ear discharging thick caseous material. At the operation carious bone was removed, but the child died after repeated bleedings from the sinus. The autopsy revealed a large gumma involving the petrous pyramid. Gummata were also present in the dura mater and in the parietal and occipital bones.

3. *Late Type of Congenital Syphilis*, in which the deafness occurs usually between the seventh and thirteenth years.

(a) *Deafness due to a Neuro Labyrinthitis spreading from the Meninges.*—Siebenmann and Mayer believe that the condition is due to a syphilitic meningitis which extends to the labyrinth. Mayer holds that this is a recurrence or an exacerbation of the condition which he has found in the internal auditory meatus of still-born syphilitic infants (*vide supra*). It is claimed in support of this view that Kretschner and Tobler have found lymphocytosis in the cerebro-spinal fluid in older cases of congenital syphilis.

(b) *Vascular Changes.*—Baratoux believes that the late form of congenital syphilitic deafness is due to changes in the vessels (endarteritis).

(c) *Otitis Media followed by Paralabyrinthitis or Invasion of the Labyrinth.*—As will be seen later, many otologists agree that the tympanic membranes are seldom normal in the late form of congenital syphilitic deafness. This points to a past attack (or attacks) of otitis media. Politzer states that congenital syphilitic disease of the labyrinth is often associated with middle-ear catarrh or suppuration. The abstractor holds that, at least in some cases, the late form of deafness is due to syphilitic otitis media—possibly with mixed infection—which either invades the bone of the labyrinth capsule, giving rise to a chronic form of osteomyelitis, or breaks through into the hollow spaces of the inner ear, causing labyrinthitis. In support of this view of the pathology the abstractor puts forward the following case, for permission to record which he is indebted to Dr. Logan Turner:—D. W., male, aged 20 years, had measles at the age of 4 years, followed by otorrhœa. The father died of aneurysm at the age of 35; the mother stated that she had had four still-born children before the birth of the patient; afterwards she had three more still-born children. At the age of 9 years the patient had interstitial keratitis and 6 months later *syphilitic ulceration of the pharynx*, followed by contraction. At this period also the boy became markedly deaf, and within a year the deafness was complete. At the age of ten years enlarged glands (syphilitic or tubercular?) were removed from the neck. The patient's speech was very indistinct, but his mother

could understand it. For some time before his admission to the Infirmary the patient had been very drowsy. Examination showed that he was emaciated. The incisor teeth were notched and the corneæ very cloudy. Syphilitic scars were noted round the knees. Wassermann reaction negative. The breathing was so noisy as to keep other patients awake at night. Only fluid food could be swallowed, on account of stenosis of the hypopharynx. The naso-pharynx was entirely shut off from the mouth and oropharynx by scar tissue. Suspension laryngoscopy revealed marked cicatricial narrowing of the upper aperture of the larynx. The patient died suddenly. Autopsy showed syphilitic changes in the skull-cap, brain membranes, lungs, heart, aorta, and liver. The brain was markedly œdematous. As an example of congenital syphilitic disease of the ear the case is not free from objection, *e.g.* Wassermann reaction negative (this is not uncommon in congenital syphilis in patients who reach 20 years), and presence of suppurative otitis media (to the abstractor this appears quite natural). Enlarged glands were removed from the neck (these also may have been syphilitic).

Microscopical examination of the middle and inner ears on both sides showed the same conditions—purulent otitis media, with great thickening of the submucous tissues; ankylosis of the head of the malleus to the outer attic wall; invasion of the marrow spaces surrounding the bony capsule of the labyrinth by a chronic form of osteomyelitis, with numerous giant cells but *no caseation*; marked erosion of the bony labyrinth capsule by the osteoclastic marrow, invasion of the semicircular canals by erosion of their bony walls and formation of granulation tissue in the perilymph spaces in such a way as to occlude the perilymph and endolymph spaces; marked dilatation of other parts of the membranous labyrinth, utricle, sacculi, and membranous cochlea. The two divisions of the eighth nerve, along with the facial nerve, were normal.

The abstractor also claims that Walker Downie's classical case supports this last view (?) of the pathology of congenital syphilitic deafness. Downie's patient became deaf at the age of eleven years. The deafness was accompanied by severe pain in both ears, though there was never any discharge. (All cases of otitis media do not suppurate and discharge.—Abs.) The deafness became complete in six months. Otoscopy showed the drumheads to be indrawn and opaque (as if from old otitis media.—Abs.).

The patient died from meningo-encephalitis resulting from a fungating gumma of the right parietal bone. Macroscopic examination of the ears showed that the seventh and eighth nerves were healthy. The middle ears were normal (at the time of death). The semicircular canals were obliterated by new bone formation and the vestibule largely filled up. The modiolus of the cochlea was thickened.

These findings appear to correspond to a stage of sclerosis and ossification following an attack of labyrinthitis.

B. *Clinical Aspect of the "Late" Form of Congenital Syphilitic Deafness.*

This is not the place to enter in detail into the symptoms and signs of congenital syphilis, but attention may be called to the wizened appearance of the child at birth, the presence of skin lesions, enlargement of the liver, spleen, and lymphatic glands, nasal catarrh (snuffles) Adenopathy, and disease of the long bones, as also of those of the face and head. Headache is common, and it is stated that the presence of severe nocturnal headaches in a child should suggest congenital syphilis.

Hutchinson, in 1861, first described the three cardinal symptoms and signs of the late form of congenital syphilis—notched, peg-shaped incisor teeth, cloudy cornea, and deafness. Wanner states that the deafness usually comes on between the ages of 7 and 9 years, or between 11 and 13 years, though later cases are not uncommon. (Cheatle reports a case aged 54.) Females are affected much more frequently than males (15 to 6), whereas in other forms of ear disease the proportion is 6 males to 4 females. As a rule the eye trouble (interstitial keratitis) comes on three or four years before the deafness. Cheatle states that choroiditis or iritis may be present though the cornea are clear. Sometimes there is a recurrence of the eye trouble with the onset of deafness. "Hutchinson" teeth are present in 50 per cent. of cases.

The deafness may come on gradually or suddenly (apoplectiform type). In some cases it is stated that the deafness comes on in a single night. It may or may not be accompanied by giddiness and disturbance of balancing. Stuempke states that in hereditary cases the deafness is usually gradual and bilateral, while in secondary acquired syphilis it is sudden and unilateral.

Otoscopy.—Politzer, Bezold, Wanner, and Hopmann all agree that the drumheads are very seldom normal. Of 33 cases examined by the abstractor, the tympanic membranes were only normal in 4, and of 31 cases observed by Nager only 9 were normal.

Functional Examination (Cochlear Apparatus).—As a rule the bone-conduction is greatly shortened and may be altogether absent. The watch is not heard by bone- or air-conduction. The upper tone-limit is much reduced, while the lower tone limit is normal or raised. Bezold states that both ends of the scale of hearing are contracted in most cases until, finally, only an island remains; even this may eventually disappear. Rinne's test is usually positive, *i.e.* air-conduction is better than bone-conduction, and the tuning fork, when placed on the middle of the vertex, is heard in the better ear. In a few cases, on the other hand, the deafness belongs to the middle-ear type.

Vestibular Apparatus.—Of 24 cases examined by the abstractor, the vestibular reaction to rotation and cold syringing was absent in 14, reduced in 7, and only normal in 4. Barany states that the vestibular apparatus fails to react in almost all cases of congenital syphilitic deafness. Hennebert (*La Presse Oto-laryngologique Belge*, 1909, No. 5) was the first to call attention to the presence of "compression" nystagmus in some cases of congenital syphilitic disease of the ear. Increase of the air pressure in the external meatus by means of a valveless Politzer bag causes a slow movement of the eyes to the same side, while aspiration brings about a slow movement to the opposite side. Hennebert finds that in these cases the rotation reaction is always absent while the caloric reaction is reduced. Buys and Alexander have also reported cases. Alexander thinks that Hennebert's sign is due to a change in the nerve endings, while Barany holds that it is due to excessive mobility of the stapes. The abstractor believes that his findings (*vide supra*) in the case of congenital syphilitic disease of the ear point to another explanation—the bony wall of one of the canals is eroded by inflammatory changes in the marrow, and the hollow spaces of the canal are partly filled up by syphilitic granulation tissue, so that the rotation and caloric reactions cannot be produced. The more powerful pneumatic test is, however, able to bring about an abnormal or reversed vestibular reaction.

Diagnosis.—According to Alexander, congenital syphilitic deafness must be diagnosed from the following conditions: (1) Idiopathic atrophy of the auditory nerves—a rare condition. Many supposed cases are really syphilitic. (2) Hereditary non-syphilitic nerve deafness. (3) Atypical cases of otosclerosis. Bezold advises that suspected cases should be examined for the presence of ozæna; saddle-back nose; perforation of the nasal septum; scars on the palate, pharynx, and angles of the mouth; deformity of the skull. Many cases show defective mental development and infantilism. The abstractor has found that a clinical history somewhat like the following can often be obtained from the patient's mother:—The patient is the eldest living child, but the mother had numerous miscarriages and still-births before the patient was born. These miscarriages, etc., occurred at gradually lengthened periods of pregnancy. Sometimes two or more members of the family show the typical Hutchinson triad of symptoms. Glover reports a case in which the eye and ear symptoms of congenital syphilis were found in the second generation, but as this is the only case on record it appears to be open to considerable doubt.

Fowler advises the application of Noguchi's skin test in the diagnosis of doubtful ear cases. The Wassermann reaction is, of course, an alternative. (See *Treatment*, section IV.)

III. Acquired Syphilis.

A. *External Ear.*

1. *Primary syphilis* of the outer ear is rare. Sohier Bryant has collected 32 cases of this condition. The disease is inoculated by kissing, biting, the use of infected towels, or by piercing the ear with infected instruments. Barbier records a case of phagedenic chancre due to piercing the ear. He states that Chinese barbers use the same instrument for cleaning the external meatus that they employ for removing condylomata.

2. *Secondary syphilis* affections of the external ear and meatus appear to be more common. Despres has observed 5 cases with papules in the external meatus among 1200 cases of syphilis. Fowler, Pogany, Guettich, Klauss, and Beck record cases of secondary affection of the external ear at periods of from 4 to 7 months after the primary sore. Knapp states that condylomata of the external meatus begin as red efflorescences, which gradually enlarge, and are followed by diffuse swelling of the meatal walls combined with secretion. Panse (*Pathologische Anatomie des Ohres*, 1912) says that pigmented scars and contraction may follow healing. In most instances the condition appears to be *secondary to syphilitic middle-ear suppuration*. Klauss says that condylomata may cause severe pain, while Pritchard states that they may be mistaken for epithelioma.

Treatment.—In Fowler's case an injection of salvarsan was given, and 5 days later it was found that the auricle had improved, while in a week all manifestations had gone except the scar in the tympanic membrane. Beck advises the local application of calomel, while Pogany uses white precipitate ointment or corrosive sublimate drops.

3. *Tertiary Syphilis.*—Cheatle states that the auricle and external meatus may be the seat of gummatous ulceration. McBride (*Diseases of the Throat, Nose, and Ear*, p. 490) reports a case of deep ulceration of the auricle.

B. *Middle Ear.*

1. *Primary Sore.*—West states that over 100 cases are on record of chancre at the orifice of the Eustachian tube. (These cases belonged mostly to the pre-antiseptic days, in which unsterilised vulcanite Eustachian catheters were transferred from an infected nose and naso-pharynx to a healthy one. Modern methods have rendered primary syphilis of the nose and naso-pharynx of very rare occurrence.—Abs.)

2. *Secondary Syphilis.* This is probably of much more common occurrence than is usually supposed. In the abstractor's opinion investigation of this subject would well repay the time and trouble

expended. Cheatle states that syphilitic naso-pharyngitis may spread up the Eustachian tube to the tympanum and give rise to the symptoms and signs of catarrhal otitis media. Bezold says that *if the labyrinth is affected in the secondary stage of acquired syphilis one usually finds otitis media as well.* As a rule only one ear is involved. The onset of deafness in these cases is usually rapid. Bourgeois reports a case of middle ear suppuration cured by anti-syphilitic treatment. The *onset of the otitis was not painful*, but the condition lasted 3 months. The case was cured within 10 days under specific treatment. M'Bride (p. 663) finds that in acquired syphilis the patient may become suddenly deaf at a varying period after infection. Tinnitus is marked, and giddiness may be present. The immediate cause may be sea-bathing (several cases). The drumhead is thickened, but inflation produces no improvement in hearing.

3. Tertiary Syphilis of the Middle Ear.—Politzer states that syphilitic otitis media is usually due to an affection of the naso-pharynx. The otitis may be a simple catarrh, a suppurative inflammation, or an adhesive process. The deafness is usually marked. Ulceration of the mucosa may occur, and be followed by caries and necrosis of the tympanic walls associated with facial paralysis. The diagnosis is difficult as the appearances are not characteristic, but rapid destruction of the drumhead and loss of bone-conduction are in favour of syphilis. Politzer states that syphilitic disease of the labyrinth may appear along with the otitis media.

Otitis media in the tertiary stage of syphilis appears to be of common occurrence. The affection may not be entirely due to the spirochæte of syphilis, but at any rate it appears that *the presence of syphilis alters the clinical course of otitis media.* Syphilitic disease (gumma or ulceration) of the mucous membrane of the naso-pharynx is usually present. In most cases disease of the labyrinth is combined with the otitis media (syphilitic pan-otitis). It appears that in these cases the middle ear has little power of repair, and the labyrinth but small capacity for resistance.

Gruenberg records a case of a male, aged 21, who had had syphilis for 2 years. The naso-pharynx was ulcerated. Within 6 months of infection there was nerve deafness with tinnitus. Death occurred from syphilitic cachexia. Post mortem the left ear showed serous exudate in the tympanum, with thickening of the mucosa; the drumhead was intact. The incus was embedded in inflammatory tissue and the bony wall of the promontory was thickened. The bony capsule of the cochlea showed wide marrow spaces. In the membranous labyrinth there were only slight changes—atrophy of the spiral ganglion in the basal coil. There were no syphilitic changes in the vessels.

M'Bride (*Diseases of Throat, Nose, and Ear*, p. 548) reports the case

of a middle aged man, suffering from acute otitis media, with severe pain, headache, and a copious discharge of pus. A mastoid operation was proposed. A history of syphilitic infection was obtained and iodide given. Within 3 days the pain had gone, the discharge rapidly stopped, and the membrane healed.

Ritter records a case in which there were tertiary syphilitic ulcers at the ostium of the Eustachian tube on both sides. Bilateral catarrhal otitis media was also present. Bruehl reports a case of a male, aged 35, who had contracted syphilis 16 years before. The patient suffered from severe headaches, tinnitus, and deafness, followed later by facial paralysis, giddiness, and nystagmus. The right ear showed catarrhal otitis. A tender swelling formed on the mastoid, but under treatment with mercury and iodide the swelling disappeared in 14 days. The giddiness and tinnitus improved, but the deafness remained.

Dan M'Kenzie records a case of sudden bilateral deafness in a tertiary syphilitic while the patient was under treatment by mercury and iodide. The patient was suffering from gumma of the cervical glands, which were excised on a diagnosis of tuberculosis. The wound healed well but the gland trouble recurred. Brunk has demonstrated a case of gumma of the mastoid accompanied by syphilitic affection of the floor of the nose. Braun had a case of syphilitic infection in a male, aged 31, who had undergone mercurial treatment at the time of the chancre 6 years before. Braun found a gumma of the right inferior turbinal and right tonsil. The drumhead on this side showed evidence of catarrhal otitis media. Deafness and tinnitus came on rapidly and the bone-conduction was shortened. Vestibular reaction present. Salvarsan caused rapid improvement.

Lueders reports 3 cases of tertiary syphilis in which the middle ears were affected. As usual the labyrinth was involved in all, while in one the facial nerve was paralysed. Specific treatment resulted in the cure of the middle ear condition, but the deafness remained. Beck has shown a case of gumma of the middle ear in a patient who had syphilis 20 years before. Cheatle describes 3 cases of gumma of the naso pharynx with deafness due to Eustachian obstruction.

Pogany comes to the conclusion that, in diseases of the middle ear, if the bone conduction is greatly shortened, we must suspect syphilis. He records 2 illustrative cases.

Barany has recorded a case of Hennebert's sign (*vide supra*) in a case of acquired syphilis. For 10 years the patient had suffered from deafness and tinnitus in the right ear with nystagmus. Movements of the head, however, caused no giddiness. The rotation reaction was normal but the caloric reaction was diminished. Compression brought about slight movements of the eyes along with giddiness.

It will thus be seen that in tertiary syphilis of the middle ear the labyrinth is almost always involved.

C. *Labyrinth and Eighth Nerve—"Neuro-Labyrinthitis."*

In the present state of our knowledge it appears to the abstractor to be impossible to distinguish clinically between syphilitic diseases of the labyrinth and those of the eighth nerve. It is true that in affections of the labyrinth alone the galvanic reaction is retained, while in those of the eighth nerve it is lost; but data on this point are given in so few instances that, for the present, cases must be grouped under the title of "neuro-labyrinthitis."

Beck (*Munch. med. Wochenschr.*, 1913, p. 2778) finds that in 80 per cent. of syphilitic patients the bone-conduction is more or less shortened although the hearing is normal. Beck attributes this to raised intracranial pressure, as he finds the same condition in cases of brain tumour and hydrocephalus. Beck performed lumbar puncture on some of his syphilitic cases and found that after this the shortening of the bone-conduction disappeared for a time, though it returned in two or three days. Cases in which the primary sore is still present and the Wassermann reaction is negative do not show this shortened bone-conduction. The latter only appears with the constitutional symptoms. The sign may be of value in the diagnosis of syphilis.

1. *Secondary Syphilis.*—Frey states that cases of neuro-labyrinthitis in secondary syphilis are much more common than is usually supposed, and claims that the work of Moriac in 1879 proves this. Habermann, in 1896, reported 66 cases of disease of the acoustic nerve, of which 34 occurred in the secondary stage. Mayer has collected 65 cases occurring since 1896, and of these 13 belonged to the secondary stage. Labyrinth symptoms, *viz.* tinnitus, deafness, giddiness, nausea, vomiting, disturbance of balancing, and vestibular nystagmus have occurred as early as the seventh day after the appearance of the primary sore (Politzer, quoted by Pogany, *M. f. O.*, 1913, p. 1333). Mayer states that the trouble may come on three weeks after the appearance of the primary sore. At the time of onset of the first skin rash neuro-labyrinthitis is by no means uncommon. The pathology of the condition is unknown, but Stuempke holds that the membranous labyrinth is involved by a syphilitic infiltration.

Beck has examined 600 cases of secondary syphilis and finds that complaints of giddiness and disturbance of balancing are by no means rare. In some of these nothing abnormal is to be found in the ears or nervous system. In others there are changes in the posterior cranial fossa, as evidenced by nystagmus of central origin and cerebellar disturbance of balancing. The third group consists of vestibular cases which show alteration in the galvanic irritability of the vestibular nerve. At the onset tinnitus is present in most cases, while giddiness is noted in 50 per cent. These symptoms may be observed before the skin rash appears. In the later stages of

secondary syphilis the ear trouble almost always comes on along with a recurrence of the rash (Mayer). Disturbance of hearing is gradual and may vary in intensity; it is usually bilateral. Functional examination shows an affection of the eighth nerve. Dalsbury reports the case of a man, aged 28, who became deaf (within a few days) only four months after infection. Paracusis Iointaine was present, *i.e.* the tuning-fork was not heard on the mastoid but was heard on the knee or foot. According to Roosa, diplacusis is a symptom of syphilitic disease of the eighth nerve, and in this opinion Freytag agrees, as the paracusis is accompanied by giddiness and loss of certain tones.

Vestibular symptoms vary from slight giddiness up to vomiting and disturbance of balancing. Mayer has not observed in secondary syphilis a case of isolated disturbance of the vestibular apparatus such as has been reported in several cases after the injection of salvarsan. According to Beck, the cochlear branch of the eighth nerve is more sensitive to toxins than the vestibular, *e.g.* quinine, salicylates, arsenic. Barany, Bondy, Neumann, and Beck have, however, observed cases of isolated loss of conduction of the vestibular apparatus in the secondary stage of syphilis without "606." Neuro-labyrinthitis may occur whilst the patient is undergoing mercurial treatment, as is shown by the case of Neumann, whose patient woke up one night after his fifteenth mercurial injection with marked tinnitus in the right ear, soon followed by giddiness, vomiting, and cold sweats; the vestibular reaction was diminished. After the seventeenth injection there was sudden abducens paralysis on the same side. Salvarsan was given, but proved useless.

The *prognosis* appears to be better in the neuro-labyrinthitis of secondary syphilis than the form which occurs in the tertiary stage.

Treatment.—Alexander and others hold that it is dangerous to give salvarsan alone if there be any affection of the auditory nerve apparatus. Mercurial treatment should always be combined with "606." Citelli reports three cases of neuro-labyrinthitis in recent syphilis in which salvarsan acted promptly after other remedies had failed. Lowinski has observed a patient who suffered from sudden occipital headache, followed in two days by facial paralysis (primary sore still present). Wassermann reaction +. Five days after an injection of salvarsan the paralysis had disappeared. Moller, on the other hand, had a case in which salvarsan failed but strenuous mercurial treatment resulted in slight improvement. Stein's patient was greatly improved by injections of salicylate of mercury.

2. *Tertiary Syphilitic "Neuro-Labyrinthitis."*—In the tertiary stage of acquired syphilis cases of neuro-labyrinthitis are not infrequently met with. In several of the recorded cases paralysis of other cranial nerves was also present, *e.g.* facial, oculomotor, glosso-pharyngeal, etc. It would appear therefore that these cases approximate closely to those

in which the eighth and other nerves are involved by gummatous infiltrations of the brain membranes at the cranial base (see later). Cheatle records the case of a man of middle age who contracted syphilis in 1894 and was treated with mercurial pills till 1900. In 1909 he suffered from pain and tingling in the right arm and right side of the face, and in 1910 suffered from tinnitus, deafness, and pain in the left ear, along with unsteady gait. Cheatle saw the case three weeks later and found the drumheads normal. Nerve deafness was present. Inunction of mercury improved the condition. Cheatle records two other similar cases. In one of these there was a gummatous mass to be felt in the naso-pharynx. West records the case of a female who suffered from an (acquired) tertiary affection of the seventh and eighth nerves. There was total deafness in one ear, with complete facial paralysis. Wassermann reaction +. The facial paralysis cleared up under mercury and iodide but the deafness remained. Harmon Smith (*New York Med. Journ.*, 17th March 1906) records a case of syphilitic necrosis of the temporal bones. Double optic neuritis was present, along with paralysis of the facial and glosso-pharyngeal nerve, deafness, and disturbance of balancing. Operation revealed necrosis reaching through the vestibule and cochlea to the apex of the petrous pyramid and backwards to the foramen magnum. Death occurred in spite of energetic anti-syphilitic treatment.

Wassermann Reaction in Nerve Deafness of Obscure Origin.—All otologists know how often they have to be content with a diagnosis of "nerve deafness of obscure origin" after they have excluded such causes as senile or arteriosclerotic changes, to the patient's occupation (boiler-makers, riveters), injury, the various infectious diseases, including influenza and cerebro-spinal epidemic meningitis, labyrinthitis following middle ear suppuration, anæmia and other blood diseases, toxic neuritis as from quinine or salicylates, etc. Such unexplained cases have been investigated as regards the Wassermann reaction by Pogany, Zange, Beck, Busch, and Arzt. In all, these observers have tested 209 cases and found the *Wassermann reaction positive in* 77, *i.e.* 36 *per cent.*

3. *Acquired Syphilitic (Gummatous) Affections of the Eighth Nerve.*—Rosenstein (*C. f. O.*, 1905, p. 165) holds that syphilitic affections of the eighth nerve are rare as compared with similar diseases of the labyrinth. The central portion of the nerve may be involved by gummata, aneurysm, or scars, while the peripheral part may be affected, along with other cranial nerves, by gummatous infiltration of the meninges and nerve sheaths. These conditions are often preceded by severe headache—sometimes of long duration. Rothmann (quoted by Rosenstein) describes the case of a male who suffered from headache, nausea, vomiting, diplopia, dysphagia, with ptosis, and loss of smell on the right side. There was also hoarseness, and pain in the left arm and leg. Examination revealed optic atrophy and paralysis

of the third, fourth, fifth, sixth, seventh (paresis), and eighth nerves. The other cranial nerves were also involved, as shown by paresis of the palate, disturbance of taste, mastication and swallowing. The recurrent laryngeal nerve was paralysed, but the pulse and respiration were not disturbed. All three cranial fosse were evidently affected. The patient recovered under mercurial inunction. It is noteworthy that both ears showed purulent otitis media, and Rosenstein states that it is by no means uncommon to find this combination of otitis media with multiple paralysis of the cranial nerves. Similar cases are recorded by Mendel and Rosenstein. Kohner's case (quoted by Rosenstein) was that of a man, aged 36, who had had severe headache and depression for two years. There was hyperæsthesia of the face and of hearing. Later, sudden paralysis of the swallowing apparatus occurred. The autopsy revealed *exostoses of various forms and sizes at the base of the skull; the brain membranes were thick and cicatricial.*

4. *Affections of the Eighth Nerve in Tabes.*—Ramsay Hunt describes five cases of otalgia due to tabes. In one case degenerative changes were found post-mortem in the pars intermedia of Wrisberg. Cheatle has observed one case of this kind. Haug (*Die Krankheiten des Ohres, etc.*, 1893) reports a tabetic case in which the acoustic nerve and ganglion were affected. Beck states that an affection of the cochlear nerve may be an early symptom of tabes. He reports the following case: Male, aged 34, had syphilis at the age of 22, and was treated by inunction of mercury. Severe headaches at times.—Wassermann reaction +. In the last five years the patient had suffered from deafness and tinnitus in the left ear, and of late the right ear had also been affected. The middle ears were normal. The left ear was quite deaf and the right could only hear a loud noise. Rombergism present. Vestibular reactions normal. Mercurial inunctions were of no benefit.

IV. Treatment of Syphilitic Affections of the Ear.

Treatment.—Such questions as that of making syphilis a notifiable disease and of the careful regulation of the marriage of syphilitic persons do not come within the scope of the present *resumé*. Nevertheless it remains true that the prevention of syphilitic deafness would be much more satisfactory than the more or less unsuccessful attempts to cure syphilitic affections of the ear once they have occurred. Much was hoped for at one time from salvarsan, but this remedy has proved very disappointing in the treatment of ear syphilis—indeed many, as will be seen later, regard it as a frequent source of ear troubles. Mott (*Brit. Med. Journ.*, 1915, vol. i. p. 192) points out that, as the normal cerebro-spinal fluid contains no protein or leucocytes, spirochætes find in it a safe retreat, and are with great difficulty eliminated, because mercury and arsenic do not pass through the choroid plexus.

With regard to *congenital syphilis*, Findlay and Robertson (*Glasgow Med. Journ.*, 1914, vol. ii. p. 401) hold that antenatal treatment gives the best results. They have treated seven pregnant women suffering from syphilis with salvarsan combined with mercurial inunction. None of the babies showed evidence of syphilis and in no case was pregnancy interrupted. Boardman (*Journ. Cutan. Dis.*, 1914, p. 545) states that congenital syphilitic infants should be treated for three or four years by means of mercurial inunction combined with neo-salvarsan. The latter is injected into the external jugular or scalp veins at intervals of two or three weeks (dose 0·015 gr. per kilo of body weight).

One of the great difficulties in treating cases of congenital syphilitic ear disease is the fact that *children are seldom brought when the deafness first comes on*. As a rule a period of months or even years elapses—at least in hospital work—before the cases are seen. If the condition were at once diagnosed by the patient's doctor the prognosis might be somewhat better. Ordinary anti-syphilitic treatment appears to be of little value, for several cases are on record in which the deafness appeared while the child was undergoing mercurial treatment on account of the eye trouble. Wanner, however, advises inunction of mercury combined with the administration of potassium iodide. Cheatle states that if thyroid extract be given along with mercury and iodide a better result may be obtained. Alexander favours the use of mercurial injections followed by salvarsan. Belam reports a case benefited by mercurial inunction, but states that the deafness is apt to recur. Bezold holds that iodide is of no value. Dench has found that in some cases injections of pilocarpine are of great benefit. Pritchard believes in local blistering. Politzer says that we should try pilocarpine first of all, while iodide and mercurial inunctions should be used later. M'Bride (p. 694) records a case of deafness due to congenital syphilis which cleared up, apparently, under treatment with pilocarpine, so that a stranger could not tell that the patient was deaf. The only trouble that remained was that melodies seemed discordant or painful. Siebenmann has seen great improvement after the administration of salvarsan, but Scheibe does not agree with this statement. So far as the abstractor knows, no cases have been reported in which the new remedies "intramine" and "ferrivine" have been used in syphilitic disease of the ear. On the whole, it must be admitted that treatment appears to be of little value. It is very important, however, that the speech should be retained, and, for this reason, the child should, as soon as possible, be admitted to a deaf-mute school. Unless this be done the speech will rapidly deteriorate, so that within a few months in the case of younger children—up to a year in the case of older children—the speech cannot be understood or may be altogether lost.

In the *treatment of acquired syphilitic ear diseases*, Stuempke (*C. f. O.*,

1909, p. 450) advises the use of mercury as soon as possible. He records two cases of secondary syphilis in which this drug caused a rapid improvement, not only in the vestibular but also in the cochlear condition. Collins (*Brooklyn Med. Journ.*, November 1905) reports six cases of labyrinth syphilis which rapidly improved under mercury and pilocarpine. Beck (*C. f. O.*, 1911, *passim*) states that mercury is the best remedy in early syphilitic lesions of the eighth nerve, he uses inunctions or injections. Valy (*M. f. O.*, 1914, p. 269) holds that mercury and potassium iodide are indicated, not only in the secondary but also in the tertiary stages. Gnastoni (*C. f. O.*, 1908, p. 531) has obtained good results from injections of corrosive sublimate into the veins according to the method of von Baccelli. Braun (*C. f. O.*, 1911, p. 297) records a case of syphilitic infection of the inner ear cured by salvarsan (*vide supra*). The methods of Swift and Ellis and of Ogilvie of introducing salvarsanised serum into the spinal canal were recently described by Eason in an abstract in the *Journal*. So far as the present abstractor knows, these methods have not hitherto been employed in dealing with syphilitic neuro-labyrinthitis.

V. Salvarsan and the Eighth Nerve.

Since the introduction of salvarsan attention has been called to the frequency of cases of "nerve relapse" after the injection of the remedy. In many of these the eighth nerve is involved alone, or along with others. As we have already seen in an earlier part of this *resumé*, cases of neuro-labyrinthitis are by no means rare in the secondary stage of syphilis, quite apart from injections of salvarsan. The question then is—Have such cases become more frequent than they used to be in the pre-salvarsan era? It is by no means easy to give a confident answer. Some authorities, such as Alexander and Bruehl, hold that syphilitic neuritis of the eighth nerve was rare in the early stages of syphilis before the introduction of "606." They believe that the salvarsan is to blame. Ehrlich, Benario and Frey, on the other hand, maintain that the nerve trouble is due to syphilis and not to the remedy. Ehrlich contends that salvarsan produces a sterilisation through the blood, except in some of the osseous canals through which the cerebral nerves pass. Here the circulation being sluggish, a few spirochætæ escape the action of the remedy, and proliferate, causing an inflammation and consequent swelling, with pressure upon the nerve, thus interfering with its function. Nichols (*Journ. Amer. Med. Assoc.*, 2nd March 1912) suggests that after salvarsan, which kills most of the spirochætæ at once, no natural resistance is established so that a focus in the nervous system, which has escaped the salvarsan, can develop unopposed. The best way to prevent nerve involvement is to administer mercury and potassium iodide along with salvarsan, and to repeat the injections as often as necessary.

Frequency of Nerve Relapse after Salvarsan.—Statistics on this point vary very greatly. Finger, Kren, Biehl, and Frey have (together) observed only 6 cases of nerve relapse affecting the eighth nerve in 732 cases injected with salvarsan. Benario has collected 62 cases of eighth nerve affection out of 14,000 treated with salvarsan. On the other hand, Zeisl and Botella (together) have noted 28 cases out of 266 patients injected. Botella thinks that these lesions of the auditory nerve apparatus may be due to—(1) Herxheimer reaction; (2) nerve relapse; (3) increase of an existing defect; or (4) arsenical neuritis.

From the clinical point of view the cases may be divided into two groups—(*A*) early, and (*B*) late.

A. *Early Cases.*—The trouble may come on within a few hours—up to a day—after injection. This form is usually regarded as an example of Herxheimer reaction due to swelling of the nerve in the narrow bony canal of the internal auditory meatus. (The Herxheimer reaction is said to occur where spirochætæ are present, or where syphilitic processes are going on.) The isolated affection of the vestibular apparatus is characteristic of this form of the affection. Rapid and complete recovery appears to be the rule.

Beck records the following illustrative case:—A girl of ten years was infected extragenitally by her sister who had syphilis. Salvarsan was given intravenously, and six hours afterwards there was marked giddiness and nausea, with loss of vestibular reaction on one side. By next day the condition had cleared up. Other similar cases are reported by Beck, Finger, and others.

B. The *late form* occurs from four weeks to six months after the injection. In several of the reported cases otitis media was present. Beck (*Munch. med. Wochenschr.*, 1911, p. 2217) records the case of a male, aged 28, who got an intravenous injection of salvarsan at the time of the first skin rash. Six weeks later there was marked tinnitus and deafness in both ears. The vestibular apparatus showed loss of reaction. Beck states that arsenic was found in the urine four months after the injection of salvarsan. The case was treated with salicylate of mercury. The vestibular apparatus recovered, but the deafness remained. Somewhat similar cases are recorded by Kinck (*M. f. O.*, 1911), Neumann (*C. f. O.*, 1912, p. 124), and Rimini (*Deut. med. Wochenschr.*, 1913, No. 2, p. 71). Neumann's case is of special interest:—Male, aged 20, was injected with salvarsan. Four weeks later tinnitus was present and the gait was uncertain; high tones lost, but other tests normal. Three months later the symptoms recurred. Rotation reaction lost; caloric reaction diminished; galvanic reaction normal. Neumann draws the conclusion that the retrolabyrinthine portion of the nerve was normal because the galvanic reaction was present. The endolymph apparatus was also normal, because the caloric reaction was present. The loss of the rotation reaction Neumann

explains by a lesion of the first neurone, sufficient to do away with the physiological stimulus (rotation), but not enough to abolish the irritability for artificial stimuli, *e.g.* cold syringing and galvanism.

It seems quite possible that both factors—syphilis and salvarsan—may be to blame, as both are nerve poisons.

To summarise the arguments *against the view that salvarsan is the cause of the ear trouble*.—(1) Cases of neuro-labyrinthitis were common during the stage of secondary syphilis before the days of salvarsan (Mauriac, Paris, 1879), and are not infrequently met with at the present time in cases in which "606" has not been employed. (2) Similar attacks are recorded during treatment by mercury and iodides. (3) Examination of the cerebro-spinal fluid in cases of "nerve relapse" appears to show that the condition is due to syphilitic meningitis (Voss, *C. f. O.*, 1913, p. 291). When the optic nerve is affected the changes appear to be inflammatory (Schanz and Tobias). (4) Cases in which salvarsan is vigorously pushed suffer less than those which receive a single dose (Ehrlich). (5) Syphilitic affections of the inner ear may be improved by the injection of salvarsan (Lange, *C. f. O.*, 1912, p. 277). (6) In the early stages of syphilis an endotoxin is formed which is liable to injure the acoustic, optic, and facial nerves or their terminations (Willcutt, *Journ. Amer. Med. Assoc.*, 1915, vol. ii. p. 602). (7) Long interval between the injection of "606" and the appearance of the labyrinth symptom, at least in some cases.

In Favour of the View that Salvarsan is the Cause of the Ear Trouble.—(1) Deafness has followed the use of salvarsan in a non-syphilitic case (lichen ruber) quoted by Lange (*C. f. O.*, 1912, p. 277). (2) Meningitic changes occur in the cerebro-spinal fluid after the injection of salvarsan, though before the injection the fluid was normal (Kinck, *C. f. O.*, 1913, p. 292). (3) Arsenic in large doses is a nerve poison (Evely, Pogany, *M. f. O.*, 1913, p. 1333), and the eighth nerve is a "locus minoris resistentiæ," especially in cases in which it is already affected by syphilis. (4) Japanese waltzing mice suffer from a congenital lesion of the vestibular nerve. Ordinary white mice, when injected with arsacetin, become waltzing mice. Subsequent microscopic examination shows degeneration of the vestibular nerves (Rothig). (5) Alexander states that he saw fewer cases of labyrinth disease in syphilis during the seven years prior to the introduction of salvarsan than in four months after its introduction (*C. f. O.*, 1911, p. 291). (6) Failure of further doses of salvarsan to effect a cure of the condition. (7) Analogy between the effects of atoxyl on the optic nerve and salvarsan on the auditory.

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CLINICAL RECORDS.

A CASE OF EXCESSIVE LOCAL OEDEMA FOLLOWING CANCER OF THE BREAST.

By DAVID M. GREIG, C.M., F.R.C.S.(Edin.).

THE unfortunate subject of this condition was a married woman, aged 38, who was referred to me in 1910 suffering from carcinoma of the right breast which had then been present two years. The case was an unsuitable one for cure by operation, as the tumour, situated in the upper quadrant of the breast, was of considerable size, and was associated with palpable glands in the axilla and in the supraclavicular region. The patient's anxiety to have something done overcame my scruples, and as extensive an operation as possible was performed. The breast was excised through suitable incisions arranged as widely clear as could be from any point of infection, and both pectorals were taken away with the breast, so as to facilitate the thorough removal of glands right up to the apex of the axilla. An angular incision was made in the neck, one limb extending along the clavicle and the other upwards along the sternomastoid muscle, and the areolar tissue and glands dissected downwards from the upper third of the sternomastoid muscle, clearing the posterior triangle and becoming continuous behind the clavicle with the axillary part of the dissection. There was a good deal of shock after the operation. The wounds healed by first intention, and she was discharged from hospital on the twenty-first day. The relief was not a long one, for five months later the thoracic wall of the axilla was studded with nodules like small shot, and more were palpable than visible. There was a good deal of induration and some blue discoloration of the skin in the supraclavicular area. I did not see her again until six months had elapsed, and during that time she has been subjected to "a course" of exposure to X-rays. The condition was distressing. The left breast had become involved and there were many glands in that axilla. The nodules in the skin extended as high in the neck as the mastoid process, and were very numerous over the chest. Pain had not been a feature throughout, but was either brought about or greatly aggravated by the extensive X-ray burn, and there was considerable swelling of the arm and less of

the forearm and hand. Seven months later, eighteen months after the operation, the condition was even more exaggerated. The carcinoma had extended, while, starting from the X-ray burn, the whole extremity had become red and eczematous and exuded a copious serous discharge. During the next three or four months the swelling of the upper extremity increased, until it assumed the enormous proportions shown in the photograph. Any photographer knows how by placing the camera the part nearest the lens will appear enlarged out of proportion to the more distant parts, but in this case no such trick was employed, and the fingers of the left hand were purposely brought in front of the right wrist, so that being nearer the camera than any other part they would bear the brunt of enlargement. The patient died two years after the operation and four years from the commencement of the growth in the breast. Post mortem showed both pleurae to be involved. There was some fluid in the left sac while old adhesions in the right had obliterated that pleural cavity. There were copious nodules in the omentum, both ovaries were cancerous, and there was an unusual carcinomatous pericarditis with little effusion.

Œdema after extensive operation for cancer of the breast is a usual occurrence. It is found more or less in evidence on the inner and back part of the arm and may not extend below the elbow. It is, without doubt, the result of the copious interference with the lymphatic circulation brought about by the removal of so many lymphatic glands and vessels. It varies with the needs of circulation, that is to say, with exercise, and is compatible with comfort and ability. If no recurrence of the carcinoma takes place, the œdema improves after some months on the establishment of efficient collateral lymphatic circulation. A more marked degree of œdema indicates something unusual and generally points to a recurrence of the disease. The œdema may be aggravated by the compression of the veins in the axilla, brought about by contraction of the cicatricial tissue. When this is the case there is prominence or dilatation of the superficial cutaneous veins and often a duskiness of the hand, fingers, and nails —the result of partial venous stasis. The swelling and œdema are less in the morning after the night's rest, becoming exaggerated with exercise during the day. The limb feels full, distended and heavy, and is colder than its fellow.

One might speak of the œdema following the operation for cancer of the breast as being primary or secondary, the former due to the immediate interference with the lymphatic circulation,

[illegible]

and the latter to cicatricial contraction. They are both perfectly familiar to all medical practitioners. Some years ago the enthusiasm of radiographers caused a pretty widespread hope that in X-rays was to be found a treatment for inoperable carcinoma. Experience has shown that this hope has unfortunately not been realised, and that there is no evidence that X-rays exercise any beneficial influence by specific action on cancer germs or cells, but that in some cases such treatment retarded growth by producing a fibrosis. It is the counterpart of what takes place in Nature, where there is a "reaction" of the tissues to the irritant of the cancer growth. It closes more or less the channels through which permeation from the primary focus can take place. In other words, it converts the disease to the so-called atrophic form of carcinoma. This explains the occasional, if temporary, success, but in the immense majority of cases X-ray treatment fails miserably. In the case I have just described the œdema was extraordinary. It was due primarily to the very extensive removal of lymphatic vessels and glands during the operation, secondarily to the cicatricial contraction which would necessarily follow such an extensive dissection, but thirdly and chiefly to the fibrosis produced through the exposure to X-rays and the severity of the X-ray burn, with the chronic septic eczematous inflammation caused thereby.

There was no question of relief by lymphangioplasty. This operation must be held to be still on its trial. To attempt it, it must be possible to gain the subcutaneous tissue which is to be drained through an aseptic area, and a *sine qui non* for success is that the affected tissue can be drained into a healthy area. Neither of these desiderata existed here. There was no part of the skin from the shoulder to the dorsum of the hand which was not inflamed and eczematous, and there was no area of healthy subcutaneous tissue with normal lymphatic circulation into which to drain.

In some cases the pain caused by the distension of œdema, the weight of the limb, and the increasing cicatricial contraction in the field of operation can be relieved by the removal of the limb. In such, a disarticulation at the shoulder is less likely to be followed by a decided benefit than removal of the entire extremity, including the scapula and clavicle. Out of 250 consecutive cases of carcinoma of the breast I have only had occasion to perform this operation once. The cases most suitable are the chronic ones, where, though there is no doubt of the recurrence, the disease has

assumed the atrophic variety and there is little carcinoma in proportion to the fibrosis. The operation cannot be thought of unless clean flaps can be obtained to cover the raw surfaces easily. In the case I have just illustrated there was much fibrosis, but chiefly an X-ray and septic change, but there was also much carcinoma widely spread, and no flaps were obtainable.

THREE CASES OF BERIBERI.

By ROBERT A. FLEMING, M.D.,
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and
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We are indebted to Major Marshall, I.M.S., for recommending these three cases to our care.

Case I.—Pedro O., æt. 24, marine fireman, *S.S. Peruna*, native of Rio de Janeiro, was admitted to Ward 22 on 12th February 1916, complaining of sore legs, pains throughout the body; duration 3 weeks. This man has been a marine fireman for 9 years, but only joined the *Peruna* on 8th November 1915, and sailed from Rio on 11th November. His home conditions do not appear to be of the best, but it is difficult to make out much about his history. His previous illnesses include only venereal disease, gonorrhœa, and soft sores.

His present illness began three weeks before admission without any apparent cause. He had pains in his legs and throughout his body, which came on gradually, and two weeks before admission he had to take to bed. He does not think that there was any definite swelling of the legs or feet prior to admission. During the preceding week the pains in his legs continued to be so severe as to prevent sleep. He had no breathlessness and no cough.

Nervous System.—The pains in his legs are worst in the calves, especially the right. His arms have escaped completely. The calves of the legs are very flabby, leaving the shin bones prominent. He has well-marked drop-foot on the left side. He is quite unable to dorsiflex the foot, and can only move his toes slightly. There is more movement of the toes on the right side. Palpation reveals great muscular tenderness, and also tenderness along the lines of both external and internal popliteal nerves, especially on the left side. There is well-marked œdema on the inner side of the tibia. Knee-jerks and Achilles jerks are absent. Plantar reflexes cannot be elicited. The reflexes of the arms are present. The electrical reactions demonstrate reduction in faradic excitability on the right side, and complete reaction of degeneration in the muscles of the left leg below the knee.

The sensory changes are extremely slight. If anything, there is some loss of acuteness of sensation, but no actual area of definite anæsthesia.

Mentally, the patient is dull and stupid.

Circulatory System.—There is marked venous pulsation, especially towards night-time, when the pulse-rate rises considerably, and, at the same time, his temperature rises to 99 or even 100 F.

The heart shows slight dilatation, and this is more obvious when he suffers from the evening pyrexia, and then he has a distinct systolic bruit, most marked in the mitral area. The other systems present no abnormality.

As the pyrexial attacks continued every night, and were not explicable by malaria or any apparent cause, one wondered whether they might not be the result of syphilis. We gave him 20 grains of potassium iodide thrice daily, and immediately the temperature fell to normal.

About a fortnight later he suddenly developed a severe hæmoptysis, spitting a breakfastcupful of bright red blood. The potassium iodide was stopped, and we had the patient's lungs carefully examined, so far as it was possible to do so; the sputum was also examined, but was at first found to be negative.

There were two subsequent hæmoptyses, and later we discovered tubercle bacilli in his sputum, and a definite area of tuberculous consolidation developed at the right apex.

The patient has been extremely depressed mentally, and each hæmorrhage has left him in a less satisfactory condition.

Notwithstanding the pulmonary complication, his neuritis has become arrested, and the legs have very greatly improved, although the tendon responses have not returned.

Case II.—Antonio F. de L., æt. 29, Rio de Janeiro. Admitted to Ward 22, 18th February 1916, with a history of pains in the legs, œdema of legs and throat, lasting for about four weeks. This man is a fireman on board *S.S. Araguaya*. His conditions at home were only moderately comfortable. He has been a marine fireman for 14 years, and shipped on 9th October 1915 on the *Araguaya*, which sailed from Rio 24th November 1915. His previous illnesses include gonorrhœa (5 years ago) and syphilis (7 years ago).

The present illness began five weeks before admission, when his legs swelled, the swelling being specially noticeable below the knee. He had no pains in his legs, and did not give up work, but

he had a sore throat, with no particular cause for it. The pains in the legs began a week later—came on gradually, and with increasing severity. They were much worse at night, preventing sleep. During the day his walking became affected, and he was ataxic, mainly due to weakness.

Three weeks ago he had to lie up in bed. The œdema disappeared under a course of purgatives and cardiac tonics, but as he was making no progress, he was sent to my wards. He was slightly breathless on exertion before he took to bed.

Nervous System.—He suffers from pain, cramp-like in character, worse in the muscles of both calves, and at times in the thigh muscles. No other muscles of the body seem to have suffered. His calf muscles are wasted and flabby, with marked fibrillary tremor, present also in some of the muscles above the knee, such as the vasti. There is some pointing of the toes, but he is able to dorsiflex his feet fairly well. His gait is ataxic, but the ataxia, as already stated, is due to weakness. He has great muscular hyperæsthesia. There is very slight œdema over the shins.

The sensory phenomena include considerable areas of partial anæsthesia on both legs, especially on the inner aspect, as tested by touch, heat and cold, etc. There is definite delay in the recognition of touch, and sensations of heat and cold are lost over a considerable part of the anæsthetic area.

There is no change in sensation above the knees, and the left leg is more generally affected than the right, the right being only involved as regards sensory change on the inner side.

The knee-jerks and Achilles jerks are absent on both sides, the deep reflexes of the arms are present, and apparently unaltered. The organic reflexes are normal.

The electrical reactions show that there is not complete reaction of degeneration in any muscles, but a distinctly less active response to faradism, and no definite change with galvanism.

There is nothing abnormal as regards the cranial nerves. The patient is intelligent and sleeps well.

Circulatory System.—There are no subjective symptoms, except breathlessness on exertion, a phenomenon only marked before patient's admission. There is no pain. The heart is dilated, especially the left side, the apex reaching to the 6th rib, and is just external to the mid-clavicular line. The right heart is also slightly dilated.

In support of these findings there is some impurity of the first sound in the mitral area but no definite murmur. In the other

areas the heart sounds are closed. The pulse is regular in rate and rhythm. There is no dicrotism of pulse, and no abnormality as regards blood-pressure.

The *alimentary system* presents nothing abnormal, except that the patient requires a daily aperient to keep the bowels active.

Case III. Manuel A. C., æt. 38, Pernambuco, Brazil, marine fireman, *S.S. Araguez*.

This man was admitted to Ward 22 on 12th February 1916, and his complaint was chiefly weakness of the knees, lasting for a fortnight, and what he describes as "sleepiness across the bowels," lasting for a week.

For the past 16 years he has been a marine fireman, and has been 8 months on his present boat. He is a married man, but with no family. His past history includes gonorrhœa and soft sores. He has been subject all his life to constipation.

A fortnight before admission, without any apparent cause, his knees became gradually weak, so that they gave way beneath him. He had pains in his leg muscles, but these were slight, and did not keep him from sleeping when in hospital. He was 5 days in bed, and saw a doctor twice before admission to the ward. What troubled him most was a dull pain, or, as he describes it, "sleepiness" across the abdomen, which his doctor considered was due to indigestion, and for which he was given a bottle of medicine, with distinct benefit.

This man has a very slight degree of neuritis.

There was very little dilatation of the heart, and only some pain on pressure over the calf muscles, while the knee-jerks and other tendon reflexes were hardly at all affected. He was treated with tinct. of digitalis, 5 minims thrice daily, and $\frac{1}{60}$th-grain of strychnine hypodermically. There was no question of the immediate improvement of this patient under ordinary diet, with the addition of plenty of eggs and milk.

These cases have been treated much on the same lines—full diet, plenty of egg being administered, while digitalis in 10 minim doses was administered to Cases I. and II., and in smaller doses to Case III.

In an extremely short time the cardiac dilatation had disappeared, but, as one would expect, the nerves did not yield to treatment so expeditiously. Massage of the affected muscles was carried out whenever pain had ceased, because it stands to reason that where there is active inflammation of nerves, as evidenced by

pain, massage could do no good, and might do considerable harm; electricity was also applied. The chief trouble was the excessive languor of the patients, and yet inability to sleep owing to pain. This was markedly present in Case I., but in Case II. was not nearly so obvious after the first two or three days. The only case which is still (6th June 1916) under observation (Case I.), and which would have been discharged long ago had it not been for the development of pulmonary tuberculosis, still shows loss of knee-jerks, although there is now complete disappearance of tenderness, and sensation has greatly improved.

It certainly takes, even with the most careful treatment, months before complete recovery can be expected to occur. From past experience of similar cases the tedious regeneration of damaged nerve fibres is the outstanding feature of convalescence.

After a careful consideration of all the cases which one of us has seen, and some of which have been already published, there seems no doubt that no conceivable cause of beriberi could produce the clinical features of the disease except a toxine, and if the absence of some element of diet has anything to do with the production of the disease, it must be by permitting the action of such a toxine, whether due to an infective organism or fungus one cannot say, but that it cannot possibly be merely the non-existence in the diet of an element without which a neuritis must inevitably occur.

The *S.S. Peruna* and *Araguez*, from which these men were brought to hospital, are two ships belonging to a Brazilian steamship company. Each ship is about 2500 tons. Both boats were destined for an alien port with large cargoes of coffee; both were compelled by our naval patrols to come into Leith and unload a considerable amount of their cargo, which was found to be intended for an enemy country.

My late resident, who is associated with me in recording these cases, Lieut. H. J. C. Gibson, R.A.M.C., inspected the *Araguez*, and saw some of the invalids on board.

The majority of the crew, even the officers, seemed to be suffering from the very cold weather then prevalent, and to be entirely unprepared, as regards clothing, for what they were experiencing.

The crew lived in the forecastle of the ship, the accommodation being extremely small. Twelve men slept and lived in a deck-house 14 ft. 9 ins. by 16 ft. 2 ins., and 6 ft. 5 ins. in height, while another division of the same house contained 16 men, that division

being 18 ft. 8 ins. in length, by 16 ft. 2 ins. in breadth, and the same height. The bunks of the men were arranged in two tiers, and there was a small stove in the centre of each division. The atmosphere of the quarters was extremely heavy and vitiated. The floor of the deck-house was paved with tiles, and the sanitary conditions were extremely primitive, the latrine arrangements consisting of a gutter about 5 or 6 feet distant from and on either side of the forecastle.

Notwithstanding these conditions, the crew seemed to have been healthy until they came into a colder climate, and there was no history of a former epidemic of beriberi on either of the boats. Of a crew of 40 men all told, 5 were suffering from beriberi, including 3 sent to Ward 22, and 2 others who were extremely slight cases and did not require treatment.

All the men seem to have suffered from constipation, and the medical officer at the docks found that this was only relieved by the administration of strong colocynth and aloes pills. If untreated, the men on an average had only a motion every 4 or 5 days. There was no medical man accompanying either ship, nor was there any medicine chest on board, although the first mate had a few drugs which he kept for his own use, and about which he said he desired the crew to know nothing.

The food on board the *Araguez*, of which the men partook, was the following:—6.30 A.M., coffee, bread and butter; 10 A.M., breakfast, consisting of stewed meat, beefsteak, rice, beans, farina, and coffee; 4 P.M., same kind of diet; 7 P.M., tea, bread and butter.

The rice, of which we got a sample, was not highly polished, and the beans, blackish in colour, were submitted to Professor Bayley Balfour for an opinion, who in a covering letter enclosed Mr. H. F. Tagg's report:—

"I have to report anent the seeds sent by Dr. Fleming that I have failed to match them with any seeds in our collection.

"From the microscopic structure of the seeds I have formed the opinion that they are probably those of a species of *phaseolus*, and examination of material in our collections leads me to regard *phaseolus vulgaris* and *phaseolus multiflorus* as species likely to be the source of the seeds in question.

"The two species are closely related, and of both there are forms with small black seeds.

"Unfortunately I have not been able to get for comparison reliably named seed samples of the forms with small black seeds, but I have examined microscopically the seeds of other forms, and

as far as one may base conclusions on similarity of structure of related but not identical forms I think the seeds sent by Dr. Fleming should be referred to one or the other of these species.

"*Phaseolus vulgaris* and *phaseolus multiflorus* are both widely cultivated almost all over the world. Both are said to have originated in South America.

"(Signed) H. F. Tagg.

"*8th March* 1916."

The black beans are stated by the men to be got from a small shrub.

The farina is a substitute for potatoes, and is made from a kind of yam. The vegetable is dried and powdered, the starch extracted, and the remains, after again drying, is known as "farina." It is not known how old the farina was which was used on the ship, but good farina is said to remain in palatable condition for 12 months. When prepared for food it is mixed with hot water or gravy and is not cooked.

There were no complaints made by the crew with regard to the food, and during the voyage, and owing to calls at Madeira and Dover, the crew have never been without fresh vegetables and fresh meat. No dried or salted meat had been used on the voyage. Of course the supposition is that the diet to which the beriberi might be attributed was that eaten by the men *before* they sailed, and that whether this is true or not the exposure to excessive cold precipitated the attack.

As the proofs were being corrected we received a letter from Professor Bayley Balfour announcing that some of the black beans had been planted and had flowered and fruited, and that Mr. Craib, the University Lecturer on Forest Botany, had identified the plants as *phaseolus vulgaris*.

We have to acknowledge much assistance in watching and noting the progress of these cases rendered by Mr. J. T. M'Auslin, the present resident physician in Ward 22.

RECENT ADVANCES IN MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

W. T. RITCHIE, M.D., EDWIN MATTHEW, M.D., J. D. COMRIE, M.D., AND A. GOODALL, M.D.

Immobility of the Diaphragm following Pleural Exudates.

Pryor (*New York Med. Journ.*, 22nd April 1916) discusses the result of examination of 83 patients who had suffered from pleural exudate. The action of the diaphragm was studied by means of the fluoroscope. In 15 instances the comparative movement of the diaphragm on the affected side was normal, in 17 the movement was more or less restricted, and in 53 one-half of the diaphragm was completely immobile. About one out of five recovered with function unimpaired, approximately the same proportion revealed disordered or limited action, and in about 63 per cent. all motion was lost.

Empyema.—When the aggregate is divided into special groups for analysis, empyema is found to be a very serious matter as far as the attendant and subsequent damage is concerned. The accepted opinion that empyema in the adult and in the child is characterised by differences in the course, duration, and subsidence of the disease has led to separate consideration of its effects in the two classes of cases. Of the 31 cases of empyema which occurred after the fifteenth year, 24 had apparently ended favourably and 4 were open cases with drainage. The surprising total of 26 revealed complete unilateral immobility. The leaflet of the diaphragm, with a few exceptions, was found in the low position or below the mid position, as seen in quiet breathing. The phrenicocostic sinus was abolished in 34 and the phrenico-pericardial angle in 31.

The half of the diaphragm was flat except in 4 instances, in which it was notched near the costal surface. In three cases slight movement upwards during inspiration seemed to occur, but this was found to be due to the costal and abdominal effort and a twisting of the lower part of the chest. The ribs of the affected side were pulled down and inward, while the spine was curved and the ribs of the normal side drawn apart and outward. The dome or any curve was always absent. Of the remaining 5 cases, one showed limited motion, with a flattened dome, and two much restricted motion with lost dome and obscuration

of the costal space. Only 2 showed evidence of full excursion equal to the opposite side.

The two patients who escaped perceptible damage were of great interest. Both had originally a resection of two ribs, and in each about two quarts of pus were removed. As far as could be ascertained, the accumulation had been present for about a week prior to evacuation. Both had decided thickening of the costal pleura, with adhesions and apparently little lasting collapse of the lung. The diaphragm seemed to move, as perceived through the opening, but the accuracy of this observation has since been doubted. One case was a girl, aged 18, the other, a boy, aged 16. Three years later the boy suffered from a sacculated empyema on the same side. At the time of the second operation the pleura was found thickened in honeycomb form and massive adhesions were exposed to view. These two patients had very severe attacks. Experience led to predictions which proved unjustified and increased the doubt that adhesions were alone responsible for the injury to the diaphragm.

Eighteen children were examined. Their ages ranged from 2 to 11. Only two had pleurisy with effusion. One recovered free mobility, one did not.

All except one of the empyema cases ended in recovery. In this case the fluoroscope revealed an additional pocket of pus.

In 8 cases the segment of the diaphragm was absolutely immovable, in 5 motion was restricted, with imperfect dome and signs of adhesion. In 5 the movement of the diaphragm appeared to be normal. These children were examined from one month to two years after operation. Renewed scrutiny later may exhibit a more hopeful picture. It is obvious that the child does not escape the crippling effects found in the adult, but the frequency of permanent injury is not so great.

Pleurisy with Effusion.—The results of pleurisy with effusion were observed in 16 adults. Nine displayed complete immobility on the affected side; one showed slight movement of a part and loss of dome; three showed normal mobility; in ten the costal space was obliterated, and in eight the phrenico-pericardial angle was not visible. Compared with the results of empyema the outcome is more favourable.

Pleurisy with Effusion and Tuberculosis.—Twenty-one cases are reviewed. In 15 the pulmonary tuberculosis was in the incipient stage; in 5 the disease was moderately advanced; in 12 there was complete immobility of the diaphragm on the affected side; 2 had greatly restricted motion, with flatness and no dome; 2 limited movement, with slight appearance of dome; and 4 comparatively normal movement. One case of this series is specially noted. A man of 30 with fibroid phthisis of two years' duration had a history of pleurisy with a large effusion on the left side, and later a smaller effusion on

the right side. He complained of great dyspnœa, which the lung lesions seemed insufficient to explain. Fluoroscopic examination showed complete bilateral immobility with pure costal breathing. The diaphragm was in the lowest possible position, and the heart was elongated and pulled down by a stretching of the pericardial attachments.

The effects from pleural exudates associated with pulmonary tuberculosis show no marked dissimilarity from those of the simple variety. Moderate or small effusions which disappear by absorption may be followed by an immobile diaphragm. Possibly the appearance of tuberculosis of the lung following some time after the incidence of a pleurisy may be traced to the inefficient and disordered work of an adherent lung no longer aided in its work by a helpless half of the diaphragm.

Pneumothorax.—Five cases are reported. One had a pneumothorax with a small effusion of fluid which disappeared without aspiration. The diaphragm had impaired movement, with loss of dome and evidence of adhesions. Another had a small attack and recovered with good movement. An examination of three cases in which artificial pneumothorax had been induced showed complete immobility on one side. It seems probable that this is an addition to the list of untoward results which may follow this procedure.

Diaphragm on Normal Side.—Extra effort with greater excursion was noted only three times. The respiration in many cases was mainly costal and increased in frequency. In 51 of the 83 cases the excursion of the unaffected side was apparently below normal and in 24 the movement was decidedly diminished.

Condition of the Lungs and Heart.—Evidence of collapse of the lung as the result of pressure was surprisingly meagre. Only 6 cases exhibited the appearance of an airless area confined to a part of the lower lobe, and 3 of these were observed from one to two months after operation. Of the open cases with drainage 4 were marked by extensive collapse, with a connecting band of adhesions attached to the costal pleura at the site of resection. Otherwise the lung was permeable but incapacitated for its full expansion by the absence of diaphragmatic help and the unknown extent of restricting adhesion.

Displacement of the heart is already fully discussed in the literature of Röntgenology.

Cause and Prevention.—Much stress has been laid on adhesions as the responsible agency. Their fettering agency is well known, but there is room for the contention that they may not be the chief factor in producing immobility and may be formed after the diaphragm has been disabled. The common sites of origin are the phrenico-colic space and between the pericardium and the diaphragm, but it is difficult to understand how they occur while fluid is present in the pleural cavity. Their appearance after operation does not explain a fixed

diaphragm, because it is found to be immobile immediately or soon after operation. Nor will adhesions between the lung and the diaphragm take place for an indefinite time, because the diaphragm does not ascend and the compressed lung does not descend to provide contact. The opinion is advanced that adhesions play a secondary part in the immobility of the diaphragm and attack a part already partially or entirely paralysed. It is essential that the question of adhesions between the diaphragm and the abdominal organs, particularly the liver, should be investigated. Their presence would explain several phases of the problem. It has been shown that massive adhesions and great thickening may be visible through the pleural opening and yet the movements of the diaphragm be undisturbed. Attention has been called to the high expiratory position of the diaphragm in uncomplicated cases of phrenic paralysis. Obviously that sign bears a relation to entirely different conditions and causes. When paralysis is associated with effusion of pus or serum the half of the diaphragm is depressed and held in low position by pressure. Its function is abolished for days or weeks. The factors of non use and inflammatory disturbance must be considered. The diaphragm becomes thickened by exudative infiltration and, later, atrophy occurs to promote stiffening and loss of elasticity, especially in the dome. This is not merely an acute but often a chronic process. Negative pressure is altered, the aspirating power of the lung is diminished, and the abdominal movements are curtailed. Any free exudate in the pleural cavity presses upon and bathes the diaphragmatic surface. The collection forms there and remains till the last in that region, particularly in the costal sinus. The costal sinus provides a pocket for retention and infection and for organised exudate and degenerative changes in the region of the muscular attachment of the diaphragm. There is a possibility that gradual paralysis of the phrenic nerve might be induced in its course through the mediastinum along the pericardium and phrenico pericardial ligament to the diaphragm.

Treatment.—So far as empyema is concerned early recognition and drainage are imperative. Thorough drainage, with a sufficient opening and as complete discharge as possible by means of change of decubitus, coughing, and deep breathing to prevent collections in the neighbourhood of the diaphragm, and especially the costal sinus, are apparently the only means we possess. There are some objectionable features in a very low opening. It is sometimes dangerous if too near the diaphragm, and much thickening of the pleura and large bands of adhesions may result. A determination to secure drainage at the lowest regions in the back adds an element of great discomfort to the patient. The long-tried experiment of irrigation did not prove to be successful.

The chief question in dispute in the case of serous effusion is,

when should we aspirate? Waiting with the prospect of absorption beyond a certain limit and acceptance of the view that a moderate amount of effusion is a natural effort in conservatism may be a mistake. How long fluid should be allowed to remain undisturbed is a matter worthy of investigation.

Polycythæmia Vera.

This condition is sufficiently rare to make the record of a case a matter of interest, and as no satisfactory treatment is known any therapeutic measure followed by improvement is worth noting.

Friedman (*Med. Record*, 15th April 1916) records the case of a Russian Jewess, aged 46, who complained of weakness, palpitation, and giddiness. These symptoms were aggravated during her menstrual periods, but there was no apparent uterine disturbance. Her hands became blue and she felt "as if her heart were going to pieces." There was occasional vomiting, constipation, and headaches. Spleen enlarged. Gastric analysis showed no abnormality. Blood showed red corpuscles, 10,000,000; haemoglobin, 134 per cent.; leucocytes, 16,200, of which 90 per cent. were polymorphs; lymphocytes, 7·5; and eosinophils, 2. Arsenic and thyroid both aggravated the giddiness. Treatment by X-rays had no effect on the blood beyond a slight reduction of the haemoglobin percentage. The spleen was not diminished in size but the appetite improved, the giddiness disappeared, and the general condition greatly improved.

A. G.

SURGERY.

UNDER THE CHARGE OF

J. W. STRUTHERS, F.R.C.S., and D. P. D. WILKIE, F.R.C.S.

The Early Changes Observed in Shell Wounds.

Policard and Phélip (*Lyon Chirurgical*, January to February 1916) have examined microscopically and bacteriologically a large number of wounds soon after the injury in the trenches. Deep wounds were selected for the investigation, and prior to the examination the only treatment had been a superficial application of iodine. The researches mainly deal with the changes in relation to portions of infected clothing carried to the depth of the wound along with fragments of shell. By observing a large number of cases at definite periods the progressive changes occurring in infected wounds could be recorded.

The authors examined most of the cases by means of films made

from the wound, including cotton or woollen fibres along with surrounding exudate and debris. They point out that cultures are not of much assistance in recognising the predominance of a particular infection, at least in the early stages and before the flora of the wound have become established. By examining a series of films the local reaction of the tissues could be followed and the appearance and multiplication of organisms could be observed.

Up to the fifth or sixth hour after injury there is, as a rule, no change in the appearance of the tissues and usually an absence of reaction in the healthy tissues. This delayed and feeble local reaction is probably due to nervous and vascular lesions produced by the impact of the projectile. In traversing the tissues a projectile will cause rupture of capillaries and of small vessels and nerves at a distance, and thus the congestion, which is the initial change in reaction of the tissues, may be delayed.

The first evidence of a defensive reaction is the appearance of leucocytes, which become obvious in films about the fifth hour. Multiplication of organisms can be observed between the ninth and twelfth hours. The first organisms to appear are Gram-positive bacilli belonging to the malignant œdema and perfringens groups. Gram-negative bacilli appear later. For a short period only bacilli are seen, but cocci begin to appear between the sixteenth and twenty-fourth hours: the latter belong to the staphylococcal, diplococcal, and streptococcal groups. From this stage onwards a great variety of organisms is present in the wound. The organisms which flourish at an early stage have a special tendency to produce necrotic changes in the tissues.

As regards the cellular reaction, the first cells to appear are neutrophile leucocytes, in the fifth hour after injury. In very toxic cases the leucocytes undergo rapid disintegration. In other cases they are transformed more slowly into pus cells. Up to forty-eight hours it is rare to meet with living leucocytes, as shown by vital staining: in later stages living cells are common. In toxic cases the leucocytes are rapidly digested: the nucleus swells and the protoplasm becomes homogeneous and acidophile. Degeneration by pyknosis occurs only in the second or third day when a successful reaction is occurring, and it is always a favourable sign. Phagocytosis is never obvious in this class of infected wounds, and it does not appear to be the essential process.

The authors have been struck by the great difference in the reaction and amount of necrosis occurring in wounds which were apparently similar in type and severity and infected by identical portions of infected material. The clothing of soldiers on active service is infected in a very uniform manner, and when the infection proves virulent it is so probably because the local conditions are favourable and not because a specially large number of organisms has been introduced.

Local factors of importance in influencing the type of infection include the bactericidal action of the blood, as indicated by Wright: the presence or absence of oxygen in the tissues, which may favour the growth of aerobic or anaerobic organisms; and the association of different varieties of organisms.

The biochemical changes associated with damaged tissues, however, are probably more important than any of the above mentioned causes of diminished resistance of the tissues to virulent infections. The presence of disintegrating tissues favours the growth of organisms and the formation of toxines. The latter may be derived at an early stage from aseptic autolysis of the tissues as well as from bacterial sources. Progressive necrosis of the tissues is largely due to toxic actions. Organisms are sensitive to slight chemical changes in culture media, and in the case of infected wounds the medium may differ according to whether the organisms are implanted in muscles rich in carbohydrates or in fats or connective tissues rich in collagen and poor in carbohydrates. The authors believe that there is thus a distinct regional resistance to infection, and have noted, for example, that organisms multiply slowly in the fat of the buttock and that necrotic changes there are rarely produced. The quantity and quality of the damaged tissues are therefore both important factors in deciding the type of infection.

In the wounds of modern warfare severe toxæmia is much more common than general infection. From the practical point of view it is important that early steps should be taken not only to destroy organisms by antiseptics but also to remove or dissolve necrosed, damaged, or disintegrating tissues, as these form the principal culture medium for the bacterial growth and the formation of toxines. These observations indicate the necessity of thorough treatment of the wound before the first four to six hours, when multiplication of organisms occurs. Solutions of hypochlorites are specially useful in treating such wounds, because they possess a solvent action on damaged tissues as well as being powerfully antiseptic. For this reason solutions of the hypochlorites are superior to antiseptics such as formalin, which hardens the tissues, or carbolic acid, which renders the tissues unsuitable as a medium for bacterial growth, but possesses no solvent action.

The Surgical Treatment of Wounds of the Pleura and Lung.

Gasquet (*La Presse médicale*, 13th April 1916) discusses the treatment of penetrating wounds of the chest in relation to the complications which may arise, such as pneumothorax, hæmothorax, secondary hæmorrhage, and the changes associated with the presence of foreign bodies.

Pneumothorax may be partial or complete, and due either to perforation of the lung or to an opening in the chest wall. No surgical treatment is required when the pneumothorax is due to a wound of the lung, if the parietal wound is closed, as the air is gradually absorbed. In the case of pneumothorax associated with a large gaping wound in the chest there is a grave risk of pyothorax developing. Thevenot and Dumarest recommend the closure of the external wound in such cases as soon as possible. By arresting the free passage of air into the pleural cavity the lung will tend to expand again and dyspnœa will be lessened. This complete closure of the wound in the parietes should only be attempted soon after the injury and before infection of the pleural cavity is established. If an empyema develops a portion of rib can be resected later for drainage. A general infection of the pleural cavity is almost certain to follow if a large wound is left open or if a tube is inserted through the wound.

Except in rare cases, wounds of the chest wall should not be opened up. As a rule the foreign body passes in between adjacent ribs, and the apertures in the chest wall close behind it. Opening up the track of the bullet only encourages infection to spread from without inwards. Probing the wound is also dangerous. The diagnosis of a penetrating wound must be made by a consideration of the symptoms and physical signs, aided, if necessary, by the exploring needle and the use of the X-rays.

In treating cases of hæmothorax it is usual to keep the patient quiet and to wait for a natural absorption of the blood. After keeping the patient at rest for one week, Gasquet prefers in such cases to withdraw a limited amount of the blood every three or four days. The advantage of this method is to shorten the convalescence and to favour the expansion of the lung. In cases where there is intense dyspnœa, either through the effusion being bilateral, or from pressure on the heart, or when the lungs are also involved, the hæmothorax must be treated by aspiration of the fluid.

The earliest sign of infection occurring in a hæmothorax is a relative increase of the polymorph leucocytes, as noted in blood-films. At a later date the clinical signs rarely leave any room for doubt as to the presence of pus. As regards the treatment of infected hæmatoma, it may suffice, in the early stages, to aspirate the fluid but if a definite empyema is diagnosed, it is always advisable to drain the lower part of the pleural cavity after resection of a portion of rib. The drainage tubes should be short and washing out of the pleural cavity is not recommended. Secondary hæmorrhage into the pleural cavity occasionally occurs. When the symptoms are urgent it is necessary to drain the pleural cavity, the partial collapse of the lung, consequent upon the pneumothorax, being sufficient to ensure hæmostasis: if, however, in spite of this the hæmorrhage continues, a more extensive

operation is necessary to permit of the wound in the lung being stitched or packed.

A bullet or portion of shell embedded in the lung substance is usually surrounded by an area of consolidation. Such an area, whether superficial, or deep, is generally adherent to the parietal pleura, and a fistulous track may lead from the skin wound to the site of the foreign body. Occasionally, however, the lung with a metal fragment embedded remains free, and a considerable number of cases have occurred in the present war in which the foreign body left *in situ* has given rise to no trouble. If the parts are infected an intra-pulmonary abscess may form. Spontaneous evacuation of an abscess occasionally occurs by discharge of the pus through a bronchus or by a fistulous opening on the surface; surgical interference, however, is usually necessary. The indications for removal of a foreign body in the lung are: (*a*) when the projectile is lodged in a consolidated area adherent to the parietes, with or without a fistulous track; (*b*) the presence of an abscess: (*c*) persistence of temperature, dyspnœa, pain on local pressure of chest wall, or occurrence of repeated attacks of pneumonia, especially if the physical signs suggest pus formation.

If the foreign body is causing no symptoms it is better to leave it alone. Before operation the exact position and depth of the projectile should be determined by X-ray photographs. The experience gained in the present war is sufficient to prove that the removal of foreign bodies from the lungs is not such a serious procedure as was formerly believed.

The Treatment of Penetrating Wounds of the Knee-Joint.

Gayet (*Lyon Chirurgical*, January to February 1916) records the results of his experiences in treating a large number of cases of penetrating wounds of the knee-joint due to projectiles. In such cases there is little time for delay in deciding upon a line of treatment, especially if portions of shell or of clothing have entered the joint. The treatment of infected wounds of the knee-joint is handicapped by the difficulty there is in getting satisfactory drainage of the joint.

Punctured bullet wounds as a rule, remain aseptic, and the prognosis, if the limb is immobilised at once, is remarkably good.

In the case of extensive wounds due to fragments of shell, the wound should be opened up at once and the foreign body removed, the interior of the joint being thoroughly washed out at the same time with ether. It is advisable to drain the joint in this type of injury, and Gayet has obtained his best results by following the method recommended by Jaboulay. This consists in removal of the foreign body through the original wound, and the insertion of a drainage tube into the supra-patellar pouch, a special incision being made if the wound

of entrance is situated elsewhere. The limb is immobilised and dependent drainage assured by suspending the limb at an angle of 45° or even nearer the vertical. By this arrangement secretions will readily drain away from the posterior recesses of the joint.

Resection of the joint is recommended when there is a limited comminution of the condyles or articular surfaces or when bullets or portions of clothing are driven into the articular surfaces.

The object in this class of case is to obtain an ankylosed limb in good position.

When extensive wounds of the joint are complicated by comminution of bones and injury to main vessels and nerves, or if infection is already present in such cases, the limb should be amputated without delay, unless the patient is suffering from a marked degree of shock.

J. M. G.

OBSTETRICS AND GYNECOLOGY.

UNDER THE CHARGE OF

A. H. F. BARBOUR, M.D., AND J. W. BALLANTYNE, M.D.

Eclampsia.

The steady flow of articles bearing on eclampsia continues, and increases rather than decreases. In the last six parts of the *Index Medicus* no fewer than twenty contributions to the subject are enumerated, and to some of these papers it may be useful here to refer, although it must be stated that no one of them claims to have discovered the exact cause of eclampsia or its infallible means of cure.

Dr. Gordon G. Copeland gives an interesting account of eclampsia as he finds it in Canada (*Dominion Med. Monthly*, 1915, xlv. 79; *Canadian Journ. of Med. and Surg.*, 1915, xxxviii. 79). In the region of causation he makes the interesting suggestion that the so-called "epidemic" tendency which eclampsia sometimes shows may point back to an epidemic of scarlet fever occurring some years previously, affecting a large number of girls and being followed in them by nephritis; then when these girls came to marry and to pass through their first pregnancy they would be unusually susceptible to eclampsia. The suggestion is worth keeping in mind, and it may be tested in future "epidemics" of convulsions; but, of course, it presupposes that eclampsia is closely associated with a preceding nephritis, and that is not a matter upon which all are agreed. He had to deal with eight cases, and he saved all the mothers; but he states that the maternal

mortality for the whole country is about 25 per cent., whilst over 50 per cent. of the babies perish. In his series of eight cases all the fœtuses, save one (a puerperal eclampsia), perished. So far as etiology is concerned, Dr. Copeland thinks that there is a specific eclampsia poison, or group of poisons, probably of protein composition; it is probably a colloid, and consequently not easily to be removed from the system by purgation. He recognises two types of the disease—the liver form and the kidney type. The former resembles acute yellow atrophy of the liver, delayed chloroform poisoning, and phosphorus poisoning; the latter is characterised by high tension pulse, headache and epigastric pain, severe nervous symptoms, almost constant œdema, and changes in the urine (albumen, blood casts, etc.). As to prognosis, emphasis is laid upon many fits, an early date of occurrence in pregnancy, a weak and fast pulse, jaundice, and anuria as grave signs and symptoms. The treatment as carried out by Copeland in the Toronto Western Hospital consisted of the following procedures in the order in which they are enumerated:—He gave, to begin with, half a grain of morphine hypodermically, and a quarter grain later if necessary, and did not use chloroform; he prevented the patient from injuring herself (sheet, gag, lateral position, etc.); he took every precaution against sepsis, remembering the eclamptic's liability to it; he gave a hypodermoclysis of 1000 c.c. of normal saline fluid at 100° F. under, not into, the breasts; he withdrew a considerable quantity of blood by aspiration from a vein or by bleeding, and found this had sometimes a most dramatic effect; he emptied the uterus, dilating the cervix under nitrous oxide and oxygen or ether and applying forceps, or using hydrostatic bags and not hurrying the dilatation too much; he tried to eliminate the poison through the intestinal tract by enemata (*e.g.* the one-two-three enema, consisting of one part of glycerine, two of magnesium sulphate, and three of water), by washing out the stomach, and giving magnesium sulphate through the tube; he reinforced these means by elimination through the skin (hot packs) and the kidneys (diuretics); and he would use, in rare cases of anuria, decapsulation of the kidney, and, in pelvic contraction, abdominal Cæsarean section. In several cases he found an ice-cap to the head did good. Dr. Copeland condemns certain commonly used means, including croton oil, veratrum viride (which he thinks is simply adding one poison to another), pilocarpine ("the sweat it gives is frequently the death-sweat," and accouchement force (most emphatically). Inhalation of oxygen was useful.

Professor J. D. Evans of Montreal (*Canad. Med. Assoc. Journ.*, 1916, vi. 110) points out that since the precise diagnosis of eclampsia can only be made by the pathologist, that of the clinician must always be provisional: indeed, he doubts whether a diagnosis of true eclampsia can be made at all during life, whilst on the other hand he states that

very extensive and typical pathological lesions may exist without eclampsia developing, and cites an interesting case in illustration. The problem in eclampsia, as he sees it, is to gain compensation of the liver and kidneys, which are not able to cope with the toxæmia; but, unfortunately, there are at present no certain methods of ascertaining the competency of the liver and kidneys in any given case. Neither the degree of the albuminuria nor the microscopic findings on examination of the urine give much clue as to the severity of the toxæmia; and the blood pressure, although usually a valuable guide, is not free from fallacies. Evans narrates some illustrative cases in which there was a serious degree of pregnancy toxæmia, and yet the blood-pressure remained approximately normal. At the same time the height of the blood pressure is generally a valuable guide, and the danger limit should be placed at 160 mm. or at 150 if the other manifestations (headache, epigastric pain, disturbed vision, etc.) are well marked. So far as treatment is concerned, Evans thinks it is significant that the remarkably good results got by Strogonoff and Zweifel were obtained without the induction of diaphoresis. He himself thinks every case must be treated individually. In the pre-eclamptic state he relies upon eliminative and sedative treatment, trusting much to milk diet, hot baths, the copious use of fluids and purgatives, with rest in bed. If the albuminuria does not decrease, the blood-pressure fall, and the toxic symptoms disappear he induces labour. Venesection, sweating, the employment of morphine and chloral in moderate doses, with purgation and the free use of fluids are the means he uses when convulsions have actually occurred. In cases at or near term he thinks surgical methods of delivery may be undertaken, but only to save the life of the child, as such operations, unless attended with considerable hæmorrhage, seem to have but little influence in relieving the condition of the mother.

Fabre and Fouilloud Buyat (*Ann. de gynéc. et d'obstét.*, 1916, 2 ser. xii. 49) report a somewhat unusual case of pregnancy eclampsia. The patient was a primipara of 22 years who had noticed swelling of the legs and eyelids at the fifth month; at the sixth month she suffered from most of the signs and symptoms of the pre-eclamptic state, and these culminated in seven convulsions. She was brought into hospital, put upon a water diet, and given chloral hydrate. Gradually the coma passed off, no more fits occurred, the albumen disappeared, and the œdema was reabsorbed; but the fœtal heart was never heard after the time of the convulsions, and about a month later the contents of the uterus were expelled intact, the placenta showing numerous infarcts. Bleeding was not employed in this case because there was no high blood-pressure.

Rouvier (*Arch. mens. d'obstét. et de gynéc.*, 1915, ann. iv. p. 71) has a high opinion of morphine in eclampsia, and he gives it in large doses,

He begins with a hypodermic injection of 2 centigrammes (one-third of a grain), gives another centigramme in half an hour, and yet another in the next half hour. If no more fits occur in the two following hours Rouvier contents himself with injecting half a centigramme more: but if the convulsions continue he gives one centigramme every two hours until two convulsionless hours have elapsed. But he does not entirely rely upon the morphine, for he washes out the stomach with 5 or 6 litres of sterile water, and he sends up high into the intestine an enema of the same, repeated in 6 or 8 hours. The patient is first put on a water diet, and then, after the return of consciousness, she is put on a vegetarian régime which is chloride-free. Rouvier has also got good results from helmitol (urotropin). He practises lumbar puncture to detect at an early date the appearance of haemorrhage, and so to form his prognosis. He has noticed that two of the grave complications of eclampsia, viz. amnesia and mania, have been much less frequent since he used abundant bleeding and morphine given systematically and in large doses. He would not hesitate to exceed the largest total dose (14 centigrammes) which he had yet employed: some obstetricians had given as many as 25 centigrammes in twenty-four hours.

R. Moulden (*Amer. Journ. Obstet.*, 1916, lxxiii. 31) ventures to draw some deductions from the study of 100 pregnancies in private practice during a period of about ten years. He makes the startling announcement that ninety-five of the cases gave a history of faecal stasis. Physical examination nearly always showed the colon to be full of faecal contents at the first investigation, generally in the early months. There were eight instances of pre-eclamptic toxæmia, and all but one cleared up upon thorough elimination being secured by all the paths of excretion. About twenty-five had albumen in the urine at some time in the pregnancy. Moulden believes that there is a close relation between the various toxæmias of pregnancy, and he emphasises colonic stasis as the great cause. He places the matter forcibly when he says "the average pregnant woman is a potential magazine of poisonous chemical compounds, whose liver has reached its limit of saturation and whose kidneys have been excoriated by the imperfectly broken down ammonia compounds." As a consequence he regards thorough bowel-cleansing as of the greatest value in prophylaxis, and next to it he places the hot pack. Patients should be educated to consult their medical man soon after marriage, and be taught how to care for their bodies properly. So far as actual eclampsia is concerned, he feels that until prophylaxis by education and instruction of the mass of the people is rendered possible the sheet anchor in treatment will continue to be induction of labour.

Dr. William Mortimer Brown (*Amer. Journ. Obstet.*, 1916, lxxiii. 251), like the previous writer, lays stress on the great importance of prenatal supervision of midwifery cases, and doubts whether the general

practitioner has yet realised this fully. He approaches also the difficult question of the place the life of the unborn child should have in all discussions on eclampsia. He admits that the fœtal mortality in puerperal toxæmia and eclampsia is very high, the causes being intra-uterine asphyxia, premature birth, and injury during delivery. He goes very near the centre of the problem when he asks, Do the dangers of intra-uterine asphyxia under the expectant (conservative) plan of treatment offset the dangers in an operative delivery (radical plan)? Reliable statistics are not forthcoming to decide this matter, but Brown thinks that in the absence of a highly skilled obstetrician both mother and child have a better chance with conservative rather than radical treatment. This is a most important statement, and it will be a matter of deep regret if its correctness cannot be either proved or disproved, for on that will depend much of the treatment of eclampsia in these days of a falling birth rate, and in the future. Brown adds another weighty statement when he says, "If the mother is in such a condition that delivery is imperative I apprehend that, in a large proportion of cases, she will not survive the delivery no matter how it is accomplished." In the treatment an efficient elimination of the toxines should be the first end aimed at, and catharsis, hot packs, colon irrigation, and bleeding are all of great value when taken along with the giving of water by mouth and bowel or of certain solutions intravenously. One must fit the treatment to the patient and not the patient to the treatment. Brown presents a table of 21 cases of eclampsia with a maternal mortality of 9·5 per cent. and a fœtal of 28·5. In 14 cases convulsions developed before the delivery, and in 7 after it, and 10 of the 14 antepartum cases continued to have convulsions after the labour, so that emptying of the uterus did not stop the fits in 80 per cent. Ten of the patients were delivered spontaneously and eleven by operative interference (one Cæsarean, three vaginal Cæsareans, three manual dilatations, two low and one medium forceps, and one bag dilatation with forceps). One mother died after spontaneous and one after operative delivery (manual dilatation and version). In all the cases but one, kidney manifestations were present. Hypertension was a common feature in the symptomatology, but, and this is significant, perhaps, the cases which died had a lower pressure than the others.

Eclampsia still defies the best efforts of the most skilled obstetricians: but there are not wanting signs that the long attack is nearing a successful close. A better knowledge of the nature of pregnancy, greater care of the pregnant woman, and active measures in the pre-eclamptic state may give a prevention, which will be better than a cure.

J. W. B.

PATHOLOGY.

UNDER THE CHARGE OF

THEODORE SHENNAN, M.D., AND JAMES MILLER, M.D.

Cerebro-Spinal Fever.

One of the most interesting problems which have become prominent during the last two years—one which the experience gathered during the war may do much to elucidate—is that of the epidemiology of cerebro-spinal meningitis.

Epidemics of the disease have been of frequent occurrence in one or other part of the world ever since the Geneva epidemic of 1885 defined the disease as a clinical entity. The discovery of sporadic cases has been a matter of more recent development since the recognition of the meningococcus as the causal agent in the disease in Vienna in 1885. Some of the great epidemics of the present century were: Portugal 1901-3, with some 3000 cases—during the course of this epidemic lumbar puncture was first used as a therapeutic measure; New York 1905, with 2755 cases, when antimeningococcal serum was first employed in the treatment of the disease; Belfast, Glasgow, and Edinburgh during 1907-8, with 725, 1238, and 138 cases respectively. In addition to these great outbreaks, isolated cases have been of almost constant occurrence in the larger centres of population. During the last two winters more or less serious epidemics have occurred, mainly in the south of England, chiefly amongst troops, and there have been a greatly increased number of the sporadic cases.

The disease shows a marked seasonal curve, the cases being almost confined to the winter and spring months, reaching a maximum about February or March and disappearing towards the end of June or beginning of July. Compton (*Journal of the Royal Army Medical Corps*, November 1915, p. 546), analysing the results in the Dorset military area during the period March to July 1915, concludes that the "cosmic factors in the determination of an outbreak are sudden saturation of the atmosphere by water vapour combined with equable conditions of temperature." The general experience is that cold wet weather predisposes to the disease. In addition and possibly underlying these factors are the further ones of overcrowding and nasopharyngeal catarrh. The meningococcus is a relatively delicate germ, difficult to grow on artificial media, and dying out rapidly unless kept at or about the temperature of the human body and in moist conditions. Colebrook and Tanner (*ibid.*, January 1916, p. 76) have some interesting observations upon the viability of the germ bearing upon the question of transmission. They found that handkerchiefs were not likely to act

as disseminating media. The germ is very rarely found in the anterior nasal secretion and very seldom in saliva. (Gordon has found that saliva exercises an inhibitory influence upon the germ.) No colonies developed in plates held under the nostrils of a carrier of the organism who was at the time breathing heavily through the nose. An explosive expiratory effort had different results. Sneezing directly on to a plate gave many colonies of meningococci. Coughing produced a somewhat similar result. Plates exposed in the sitting- and sleeping-rooms used by a group of carriers only on one occasion gave colonies of meningococci. These observers deduce from their observations that the microbe, which confines itself almost entirely to the nasopharynx, is dislodged with some difficulty.

The above observations, in conjunction with the well-known fact of the delicacy of the organism, emphasise the relationship between closeness of contact and overcrowding in the transmission of the disease. As regards the relationship between nasopharyngeal catarrh and the disease, Andrewes, Bulloch, and Hewlett, in their *Report to the Medical Research Committee on Cerebro-Spinal Fever*, 1916, after an analysis of material supplied by a number of observers in various parts of England, conclude as follows :—"While it cannot be denied, in face of some observations, that individual cases of cerebro-spinal fever may be ushered in by sore throat, the more usual relation between the disease and catarrh seems a fortuitous one, dependent on the fact that catarrhs and cerebro-spinal fever have a similar seasonal prevalence—there is the same sort of relation as there is between catarrh and the price of coal. There seems, however, some reason for the belief that chronic catarrhal conditions of the nasopharynx may be associated with the persistence of the carrier condition. It may be added that more than one of the reporters suggest excessive smoking as a cause of much of the pharyngeal catarrh seen amongst the military population" (p. 45).

It must be admitted that observers are not entirely at one regarding the relationship of the meningococcus to cerebro-spinal meningitis. Hort, Lakin, and Benians (*Journal of the Royal Army Medical Corps*, February 1916, p. 153) found that the fresh filtrate of fresh cerebro-spinal fluid from two acute cases was highly pathogenic for apes, and concluded that it was possible that the true causal agent of the disease might be a "filter-passer." By incubating the cerebro-spinal fluid overnight in addition to the meningococcus a number of other germs have been separated, notably a minute bacillus. These observers consider that the so-called meningococcus may be a late stage in history of the real infective agent. Commenting upon this work, Andrewes, Bulloch, and Hewlett say "We find such views far from convincing, and they are at variance with the experience of the overwhelming majority of bacteriologists who have studied this disease" (*loc. cit.*, p. 7).

The meningococcus being accepted as the causal agent in the disease,

and the nasopharynx being recognised as the primary focus of infection (a fact which is very generally admitted), how is it that the organism reaches this focus? It may be stated that the prevalent view is that infection takes place by a susceptible individual coming into close contact with (1) an individual suffering from the disease and still harbouring the germ in his throat, or (2) a "carrier" of the germ—in other words, an individual who is carrying the organism in his nasopharynx but who is not susceptible to it.

Scott (*Report of Medical Research Committee*, p. 30) found the meningococcus in the pharynx in 16 out of 19 acute cases of the disease. In 50 per cent. of his cases (*Local Government Board Cerebro-Spinal Reports*, 1916, p. 66) complete convalescence occurred before the nasopharynx was free. Walker Hall (*Report of Medical Research Committee*, p. 29) found the organism in six cases persisting for a period of from 19 to 41 days. It is clear, therefore, that a case of the disease may be a source of infection for others during the attack and for a variable period thereafter. He may, in fact, become a "carrier."

The possibility of the dissemination of the disease cerebro-spinal meningitis by means of individuals moving from one place to another has long been recognised, although it is only comparatively recently that the bacteriological aspect of the problem has been worked out. Foster and Gaskell (*Cerebro-Spinal Meningitis*, Cambridge University Press, 1915) quote the case of the 18th Light Infantry of the French army which, quartered in the Landes during an outbreak of the disease in 1832, visited successively Bordeaux, Rochefort, Versailles, Chartres, Metz, Nancy, and Strassburg during a period of some years, and in each place, at the usual time of year—January to March—outbreaks of cerebro-spinal fever occurred amongst the civil population. The bacteriological proof that healthy individuals harbouring the meningococcus in their nasopharynx can infect other individuals with cerebro-spinal meningitis is by no means easy. To begin with, it is a matter of very considerable difficulty to differentiate the germ from other closely allied organisms which are normal inhabitants of that region of the body. It would take us too far to go into the various points of difference between the meningococcus and organisms such as micrococcus pharyngis siccus, micrococcus catarrhalis, etc., suffice to say that the germ of cerebro-spinal fever is a fairly large coccus which occurs in pairs or masses, loses the stain with Gram's method, is easily emulsified in water, grows with some difficulty, requiring blood or blood-serum media and a temperature above 25° C. Most observers agree that it shows certain fairly constant fermentation reactions with sugar, producing acid from glucose and maltose but not from galactose, lactose, or saccharose. It shows positive agglutination with homologous anti-meningococcus serum and it has the capacity of removing or absorbing the specific agglutinins from such serum. In relation to the last two

tests it must be stated that the reaction is not given with equal intensity by all strains of meningococci. In fact, in this way at least three types of true meningococci have been distinguished by the workers at the Central Cerebro-Spinal Fever Laboratory, R. A. M. College, London. The crucial test of pathogenicity is denied us in the case of the meningococcus. There is no laboratory animal which will give us an answer to the question —Is a particular organism resembling the meningococcus actually capable of producing the disease cerebro-spinal fever? When a diphtheria-like bacillus is isolated from a throat it is possible by injecting a pure culture of the germ into a guinea-pig to get an answer to the above question. Although this most important test of all is not available in the case of the meningococcus, it is possible to attain a very great degree of certainty in the identification of such organisms. The chief point in which strains of suspicious germs vary is their sugar fermentation reactions. Compton (*Journal of Royal Army Medical Corps*, November 1915, p. 546) states that only one out of six strains of meningococci obtained from cerebro-spinal fluid gave the typical fermentation tests. Four strains failed altogether to ferment glucose. Sellars (*ibid.*, December 1915, p. 676), although he found that a majority of his strains obtained from cerebro spinal fluid were in this respect typical, records that strains obtained from the nasopharynx gave very divergent results. On the other hand, Eastwood, Griffiths, and Scott (*Local Government Board Cerebro-Spinal Reports*), working with solid media, have obtained very uniform results as regards fermentation reactions.

In spite of the above-mentioned slight variation amongst strains of meningococci derived from various sources it may legitimately be concluded that it is possible to decide with tolerable certainty whether a given organism is the germ which is so constantly found in cerebro-spinal fever or not. This organism is found in the nasopharynx in a certain proportion of healthy individuals who have not come into contact with cerebro-spinal cases. Scott found it in 13·7 per cent. of 138 non-contacts. Such individuals are meningococcus carriers. Further, as already stated, the organism persists for a variable period in persons who have recovered from the disease. The maximum period is stated by Scott to be more than three months. Andrewes (*Brit. Med. Journ.*, 13th May 1916) found that most cases remained carriers for several weeks, one for more than seventy days. It is clear, therefore, that recovered cases may act as carriers for at any rate some time. The carriers who have not suffered from an attack of the disease do not appear to be affected by the presence of the germ. They do not necessarily show signs of catarrh. Their blood, according to Colebrook and Tanner (*loc. cit.*), does not show evidence of antibodies. The process of formation of a carrier was observed by them in the case of three medical men coming into contact with cerebro-spinal cases who were periodically examined bacteriologically before and after infection.

The establishment of the microbe in them was entirely unsuspected, being unaccompanied by any change in general health or by local catarrh. They remained perfectly healthy. Up to the present time cerebro-spinal fever has not been observed to arise in a person known to be a carrier, and many of these individuals have been under observation for months. The curious thing is that in spite of the delicacy of the germ it is very difficult to get rid of from the nasopharynx. Colebrook and Tanner tried immunising the carrier by vaccine treatment, the local application of antiseptics, and the replacement of the germ by implanting other bacteria. All three methods proved ineffective. The organism, however, tends eventually to die out of its own accord.

The relation of the carrier to the disease may be taken as established. What it is that determines the type of outbreak, whether epidemic or sporadic, is a point which requires further investigation. The experience of the outbreaks in this country during the last two winters has been that only very seldom do two cases occur in the same place at the same time; in other words, we have merely experienced an increased number of sporadic cases. One might venture to suggest an explanation of the two types of the disease in this way:—The *sporadic type* is produced by a susceptible individual coming into close contact with a carrier. Fortunately susceptible people appear to be few and far between, because carriers are not infrequent. An *epidemic* arises when for some reason the virulence of the germ is raised so that a much greater proportion of individuals come into the category of the susceptible. J. M.

NEW BOOKS.

Pathological Lying, Accusation, and Swindling: A Study in Forensic Psychology. By WILLIAM HEALY, M.D., and MARY TENNEY HEALY, B.L. (Criminal Science Monograph No. 1.) Pp. xii. + 286. London: William Heinemann. 1915. Price 10s. 6d. net.

THE editors announce that this is the first of a series of monograph supplements to the *Journal of Criminal Law and Criminology*, the publication of these supplements being authorised by the American Institute of Criminal Law and Criminology. The editors have made an auspicious beginning in this first volume, which is both interesting, well written, and novel. The definition of pathological lying (which from a practical point of view is more important than pathological accusation or swindling) is "falsification entirely disproportionate to any discernible end in view, engaged in by a person who at the time of the observation cannot definitely be declared insane, feeble-minded, or epileptic." Hitherto nothing has been written in English on this malady, if malady be the correct term to apply to moral abnormality, and the literature, mainly German (one would like to suppose that pathological lying is a peculiarity of the Teuton), consists largely of reports of isolated cases. The author's work is based on a series of carefully investigated personally observed cases, and it seems to us to bear out the view that such a condition as pathological lying, in the sense defined, does exist, apart from insanity. The majority of the authors' cases were women—generally young women—who apparently belonged, for the most part, to the lower and lower-middle ranks of society. Roughly speaking, they were adventuresses—wanderers whose vagrant habits at best caused distress and suffering to their friends, and at worst led them into conflict with the law for swindling and fraud, usually of minor degree. Their habit of falsification showed itself in lying, always unblushing, often purposeless, or at least adapted to attain only the most nearly immediate object, and often contradictory. The habit of falsehood was in all cases of long duration, and underlying it there was sometimes an element of romantic imagination and a variable degree of self-deception. Sometimes the fabrications were so elaborate, numerous, and changeable that the person had apparently forgotten what in her past was real and what was imagined or invented; in other cases she seems to some slight extent to have convinced herself that her fabrications had a real foundation. Two striking features in the histories of these cases are, first, the marked

prevalence of definite neuropathic heredity ; and, second, the frequency of a bad early environment. So far as the evidence goes, these must be looked on as the predominating causes of the condition ; much less important, as it seems to us, was the sex motive (on which the authors do not lay excessive stress) and the existence of an imaginative disposition and gift for language.

Minor degrees of lying are familiar enough, but pathological lying in the form depicted by Dr. and Miss Healy's cases is far from common. Still, one can recollect hearing of persons suffering in this way. In a *cause célèbre* tried in the Scottish Courts a few years ago one of the accused was probably a pathological liar of the kind described above. As was subsequently shown, she had completely deceived her husband as to her previous history and as to many of her actions after her marriage, and only a most careful investigation after both spouses were convicted elucidated a most elaborate system of falsification extending over a course of years. It is a characteristic of some of these habitual falsificators that they have so great a faculty of appearing absolutely candid and sincere that they constantly impose upon their victims and may enjoy a long career of deceit before they are finally exposed.

When a case of this kind is detected the defence of insanity is often put forward. In the criminal case above alluded to, a number of alienists who investigated the mental condition of the female prisoner arrived at the conclusion that she was, if not insane, at least only partially responsible. Where lying is the only symptom it is difficult to show that insanity is present, and consequently difficult not to hold the person responsible from this point of view. For this reason the authors have made an important contribution to morbid psychology, for they have collected together a series of cases which form a distinct clinical type, and which certainly are not insane in the ordinary acceptation of the term. If, then, it is a fact that lying may exist as a more or less isolated pathological condition, it will not be necessary in order to recognise this form of moral defect to prove from other points of view that the patient is insane or neuropathic. Lying of this peculiar habitual, almost causeless type will be enough. In how far such persons are really irresponsible is quite another matter : they are certainly dangerous pests in most cases, though sometimes sinned against as well as sinning. They appear, however, to be more suitable for institutional treatment than confinement in prison. Unfortunately the habit seems to be almost ineradicable, and the outlook as to improvement rather poor. We consider that the authors have to a large extent made out their case for the existence of a pathological, non-insane form of lying, and that their work deserves the serious attention of jurists, alienists, and psychologists.

Cerebro-Spinal Fever. By MICHAEL FOSTER, M.A., M.D., Captain, R.A.M.C. (T.F.), and J. F. GASKELL, M.A., M.D., Captain, R.A.M.C. (T.F.). Pp. viii. + 222. With 19 Illustrations. Cambridge: At the University Press. 1916. Price 12s. 6d. net.

THE authors of this interesting volume were in charge of the cases of cerebro-spinal fever treated in the Eastern Command last year, and give a very complete clinical, pathological, and epidemiological description of the disease as it appeared in that particular outbreak. The clinical features are very carefully and minutely detailed, and a valuable chapter is devoted to the bacteriology of the meningococcus and other Gram-negative diplococci. It was a happy idea to include also a very useful and well-illustrated account of the anatomical arrangements and relationships of the membranes of the brain. An appendix gives a remarkable example of the spread of the disease by means of carriers. The plates, most of which are in colour, are quite good and illustrate admirably the rashes and other conditions which they are intended to represent.

Captains Foster and Gaskell agree with those authorities who hold relapses to be infrequent. It must be admitted that, as they say, the cause of the difference of opinion is the varying significance of the term "relapse." Perhaps the accesses of fever which interrupt the convalescence of a case might be termed more accurately "recrudescences," but we notice that the authors themselves on page 49 refer to "slight relapses," so they are not entirely free of the fault themselves. But in any case, real relapses, presenting most of the original diagnostic symptoms and associated with the return of large numbers of cocci and polymorph cells to the spinal fluid, were not at all infrequent in the Edinburgh outbreak of nine years ago.

The main interest of the book is perhaps the very able advocacy of repeated lumbar puncture as a systematic method of treatment and the argument that relief of pressure should be the real therapeutic object. The authors present a very strong case; they obtained excellent results, and claim with reason that the absence of chronic sequelæ in their own and other cases treated recently suggests that the comparative frequency of such conditions in former epidemics might well be due to the fact that pressure was not relieved by puncture. They had no success with serum, and we fear that such an experience has been only too common for the last eighteen months. The serums obtainable have certainly not been homologous to the strains of meningococci responsible for the recent outbreaks. Nor can polyvalent preparations be expected to be very successful. But, given a homologous serum, it would, we think, be more reliable than merely repeated drainage. Nevertheless the authors have done excellent work in proving what can be done without serum treatment, and their method

may be copied with great advantage. We are pleased to see they recommend the use of an anæsthetic for lumbar puncture. We agree that it is actually of benefit to many patients as a sedative, and we consider it almost brutal to dispense with it if punctures are frequently made.

Captains Foster and Gaskell have not in their cases met with the prodromal catarrhal stage described by some recent writers, and our own experience has been the same. A sudden onset, indeed, appears to be one of the most constant features of the disease in adults.

Although the value of the book is to some extent limited by the fact that it is based only on the observation of a particular class of male adults, and a not very large number of cases in all, and that therefore the results of treatment cannot be fairly compared with those of any series of cases which includes all ages, we can most confidently recommend it to all who are interested in cerebro-spinal fever. Medical officers in charge of troops at the present time should all study it. They will find it most useful in dealing with the type of illness at present existing. But epidemics vary much in type, and we should not be surprised if the authors modify some of their views if they renew their acquaintance with the disease in some future epidemic.

The Sex Complex: A Study of the Relationships of the Internal Secretions to the Female Characteristics in Health and Disease. By W. Blair Bell, B.S., M.D.(Lond.). Pp. xx. + 233. With 50 Illustrations. London: Baillière, Tindall & Cox. Price 12s. 6d.

In this interesting piece of work the author discusses what he terms the sex complex, being the relationships of the internal secretions to the female characteristics and functions in health and disease (title). He further puts in the forefront of his work the statement *propter secretiones internas totus mulier est quod est.* The term "sex complex" does not seem to us a particularly happy one, as it does not suggest the relationship the author is investigating; some such term as "sex endocrine complex" would give a more exact idea of the writer's aim.

In regard to what constitutes femininity, some attention should be paid to terminology. *Femina* is a *female*; *mulier*, a *woman*: the first suggests sex, the second an assemblage of qualities more than mere sex, and yet making up the totality of what may be termed "the feminine." We must define "sex" both in the male and female by the sex glands; to what maleness and femaleness is due is difficult to say. They may be due to the sex glands *plus* the other internal secreting organs, but one cannot leave out the psychosexual system.

In the first hundred and twenty-two pages the experimental results of removal operations on the internal secreting organs, where possible,

and of the sex organs are given and the results analysed in an interesting fashion. It would take too long to summarise these, but this part is the valuable portion of the book.

When the author takes up the practical application of his views he is not so successful.

Thus his case of "partial glandular hermaphroditism" was that of an apparent woman, 17 years of age, who was female in every respect except that she had the male arrangement of hair on the anterior abdominal wall, a deep voice, and a genital tract as in a female, except that adenomatous tissue was found in the left ovary when the internal sexual organs were explored and a piece taken from each sex gland. This was diagnosed as malignant, and a side-to-side operation performed.

Ova were found in both sex glands, but the adenomatous tissue contained no sperm-cells and was merely simple adenomatous tissue, or, really, Mullerian epithelium, as is occasionally found in the ovary of the human female. The author did not find any suprarenal hyperplasia, but this is only found in the rare female pseudhermaphrodite and not in such a case as the author's. His case is merely this—a female with some slight inversion in hair and larynx and the very rare condition of an inclusion of Müllerian epithelium in one sex gland.

The second case discussed (p. 142) is an ordinary male pseudhermaphrodite. It is remarkable that the author does not discuss inversion at all, although it is present markedly in this case. True hermaphrodism has not as yet been shown to exist, that is, no case of ovatestis has been satisfactorily demonstrated.

Although the author believes in the internal secreting organs as the cause of femininity, he is unable to apply his theory in these two cases. He bases his diagnosis of the first case on the ovary, and says nothing about the endocrine organs. If we leave out of account the suprarenal organs, the other endocrine organs, both in the male and female, are parallel in structure and function. We do not know why the hyperplasia of the suprarenals exists in the female pseudhermaphrodite, and therefore what facts can as yet be put forward to support the author's theory that *propter secretionem internam totus mulier est quod est?* The answer is, none. In many points this work is painstaking and valuable, but the theory put in the forefront of the discussion, only to be abandoned when actual cases are considered, makes part of the book appear to be the statement of a paradox followed by its abandonment.

It would be valuable if such a competent investigator as the author would take up fully this question of the relations of the simple hyperplasia of the suprarenals to female pseudhermaphrodite cases, and we hope that in a future edition he will give us the benefit of this work, and of a more rigid line of thought. The general get-up of the book and the illustrations are excellent.

The Institutional Care of the Insane in the United States and Canada. Edited by H. M. Hurd, M.D., LL.D. Vol. I. Pp. viii. + 497. With 30 Illustrations. Baltimore: The Johns Hopkins Press. 1916. No price stated.

This history of the care of the insane in America is brought out under the auspices of the American Medico-Psychological Association, which deputed a committee to undertake the task in 1908. This committee divided the subject amongst its members, and the resulting volume is a compilation of their historical researches under the editorship of Dr. Hurd. Considering the wideness of the subject and the size of the book the committee may be heartily congratulated on having produced a handy but useful and most interesting volume.

The first chapter gives a history of the origin and development of what is now known as the American Medico-Psychological Association, and its official publication, *The American Journal of Insanity*. This is natural, as the book owes its existence to their action, and still more because from the first the superintendents and medical officers of asylums who founded and still form the majority of the members of that Association took an active interest in the care of the insane of the community and in the provisions required for this care. But from the general point of view the most interesting chapter in the book will be found to be that in which an account is given of Miss Dorothea L. Dix. This remarkable New England lady, through possessing unusual natural gifts, and by combining these with unceasing labour for over twenty of the best years of her life, was the means of establishing over thirty asylums in the Eastern, Middle, and Southern States, as well as in Canada. She began her labours in 1841, and three years later the first fruits were visible in the passage of an Act establishing a hospital for the insane in the State of Massachusetts. After thirteen years of strenuous work in other States she revisited England, the immediate cause of this visit being the painful and unexpected shattering of her hopes of inducing Congress to place the subject of her efforts on a national footing. On this visit she renewed acquaintances made eighteen years before, and within a month of her arrival she resolved to visit Scotland to investigate the conditions of the insane there. After doing what she could in Edinburgh to remedy defects which undoubtedly existed she resolved to go to London. There, with the aid of Lord Shaftesbury, she so impressed her views on the authorities that a Commission was speedily appointed. As a result of its investigations the Scottish Lunacy Act of 1857 was passed, constituting a Board of Lunacy and securing reforms which have made an immense difference in the lot of the insane there. Amongst other friendships formed in this country may be mentioned that with Dr. Hack Tuke, and many other prominent specialists in England. It will thus be

seen that her work, though chiefly carried out in America, had close relations with our own country, and for that reason ought to be known and recognised here. She also proceeded to many European countries and left marks of her noble efforts in these lands as well. The chapter relating all these facts furnishes a most fascinating and stimulating record. Other chapters contain information regarding the administration and construction of asylums, research work, training schools for nurses, private care of the insane, and the laws regulating commitment and detention. It will thus be seen that the book is full of most valuable and interesting information to all those who have to deal with the care of the insane or are in any way interested in the matter.

Collected Papers on Analytical Psychology. By C. G. Jung, M.D., LL.D. Authorised Translation by Dr. Constance E. Long. Pp. xviii. + 392. London: Baillière, Tindall & Cox. 1916. Price 12s. 6d. net.

The atmosphere of this book is decidedly more respirable than that of any book we have yet read on the subject; indeed, it is possible that, if the new psychology had been introduced to English readers from the Zurich and not from the Vienna school, the opposition to it would not have been so powerful, certainly not so well founded as it has been.

While the erotic origin of the neuroses is fully maintained, its phallic aspect has been rejected. Love, in the eyes of Jung, means something more than, and even may be unconnected with, gross sexuality. It is true that most of the Anglo-American followers of Freud have said the same of him in the prefaces to their works and translations, but all students of the Freudian literature know that these promises are not fulfilled in the works themselves. Jung, with more original mind, has not yielded to the general belief that a thing is necessarily true because Freud says that it is, and he has broken away, and conceived of love as a thing essentially psychical. There are even hints of a working, as opposed to a theoretical, belief that any powerful emotion which affects the individual may cause a neurosis, as Dejerine has maintained for years. Most people would agree that of the emotions love is one of the most powerful.

There is a further capital difference between the two schools. The Vienna teaching has been that as soon as the complexes are exposed the patient is cured. Nothing further need be done. From this Jung dissents. Thus, in the interpretation of the dream, he, like Freud, discovers the latent content, but he does not end there. The dream is regarded as holding something more than its latent content. It is a parable, which not only reveals certain facts after the disguises have been removed, but also has in it a moral for the patient. This moral

must be discovered and pushed home, before the whole value of the dream has been exhausted. Our dreams, then, are one long series of Æsop's fables, which most of us read night after night without heeding the wisdom they might impart. Here we have frankly a return to the despised theory of re-education, a re-education, however, which it would be claimed follows a more thorough attempt than was made by the older psycho-therapists to discover what it was that really needed re-education.

In the matter of the technique of dream interpretation a good deal of cold water is thrown on the idea of fixed symbols. There are a few, says Jung, but not many. This seems to be an advance on the idea that most objects represent the penis; it is also more in harmony with the doctrine on which the followers of Freud have laid so much stress, that all psychology is individual.

As might be expected, the theory of the unconscious is treated very fully, and as this psychological term has so many meanings it is well that this should have been done. In analytic psychology the term connotes all those thoughts and memories which have disappeared from consciousness, and which cannot by any searching of the memory, or by deliberate thought, be again brought into consciousness, but which can be so brought by psycho-analysis. Therefore the search for the unconscious is a fundamentally different thing from an anamnesis. This is of course disputed by the opponents of psycho-analysis, who maintain that after an hour or two of conversation they receive precisely the same information which the psycho-analyst gets after employing his methods for weeks. It is certain that one doctor will be told in ten minutes intimate secrets which another would not in as many years. The latter would tend to think that the information had become unconscious in the Freudian sense. In his chapter on the association method Jung describes, with much pride, how the method detected which of three nurses had been guilty of peculation. Apart from the question as to whether ordinary interrogation would not have done as much, it is a fact that the thief was fully conscious of what she had done, for the crime was only a few hours old, and, naturally, she boggled over such words as "money," "leather," and "bag," which referred to the stolen goods. There is no trace of the unconscious here, but very much the reverse.

It cannot be said that Jung makes psycho-analysis easier to practise. He makes it more difficult, for he is more thorough and more far-reaching than Freud. He is certainly cleaner and saner.

The book has been translated by Dr. Long into pleasant, readable English, and she is to be congratulated on this and on her choice of these essays.

The Meaning of Dreams. By ISADOR H. CORIAT, M.D. Pp. 194. London: William Heinemann. 1915. Price 5s. net.

DR CORIAT certainly has one good qualification for an author, namely, that he holds a high opinion regarding the importance of his subject. "For abnormal psychology," he tells us, "dream-analysis can be compared only in importance with the discovery of the origin of species and of the factors of organic evolution in the field of biology. The analysis of dreams is not only of great theoretical value in the understanding of the unconscious, but has its practical side as well, in giving medicine the most potent instrument which it has ever possessed in the treatment of certain functional nervous disturbances."

These are strong words, but not too strong to emphasise the dictum that no physician worthy of the name can afford to neglect any means of relief in the treatment of functional disorders, seeing that these are just as productive of suffering and incapacity as organic diseases. Dr. Coriat's little book is simply written, and may be recommended as an introduction to the study of dreams by psycho-analytic methods to those who are disinclined to go straight to the great work of Freud.

Modern Medicine and Some Modern Remedies. By THOMAS BODLEY SCOTT. With a Preface by Sir LAUDER BRUNTON, Bart., F.R.S. Pp. xi. + 159. London: H. K. Lewis & Co., Ltd. 1916. Price 4s. 6d. net.

THIS book is of a type all too rare in medical literature. It is written by one long engaged in general practice, who, to the experience gained by wide and careful clinical observation, brings knowledge of contemporary scientific medicine, and of such more recent advances in physiology and pathology as bear most immediately on medicine proper.

In the clinical study of disease, while the general practitioner is in many important respects placed at a disadvantage to the consulting or hospital physician, there are other aspects of medicine in which he is in a better position to accumulate data for generalisation than the former. Thus, for example, in an interesting chapter devoted to the treatment of bronchitis and bronchial asthma, Dr. Scott says that vaccine therapy—in which he is a firm believer—is of little use in the treatment of chronic bronchial asthma if it be not continued for at least two years. One wonders how many consulting physicians have the opportunity of following up a sufficient number of such cases under vaccine treatment for two years to justify an opinion of this kind.

There are a great number of points in the book to which, did space permit, one should like to refer. But only a few can be mentioned. The author begins by emphasising the relative smallness of the information which the stethoscope, taken by itself, affords in cardiac cases as far as

prognosis and treatment are concerned, and the necessity of supplementing it by most careful observation of clinical symptoms, as well as by the use of the sphygmograph and the manometer. Many most useful notes are given regarding the value of drugs, especially those of the vaso-dilator class. But perhaps the most interesting part of the book is that in which Dr. Scott records his use—and it is a well-reasoned use—of glandular extracts. He is a convinced believer in their value. Specially suggestive are his remarks on thyroid treatment, and the wideness of field in which we may look for benefit from thyroid, both by itself and in conjunction with other extracts. "If we have an open choice of remedies, I think we should choose the one that already belongs to the animal system." Dr. Scott leans towards the more extended use of endocrines as against ordinary vegetable and mineral drugs—the "native" rather than the "alien."

Cambridge Public Health Series: Occupations from the Social, Hygienic, and Medical Points of View. By Sir Thomas Oliver, M.A., M.D., LL.D., F.R.C.P., etc. Pp. viii. + 103. Cambridge. 1916. Price 6s.

Any work from Sir Thomas Oliver is sure to claim attention, and though the present semi-popular treatise does not claim to unfold anything new, yet it contains a wealth of most interesting and valuable information. The author very wisely remarks that the prosperity of a nation is intimately bound up with the health of the people who compose it, and though factory legislation has accomplished much, much yet remains to be done to improve still further the conditions under which labour is carried on.

One would not perhaps have thought that agriculture claimed the largest amount of labour in Great Britain, yet such is the fact. Of 15,388,501 occupied persons in 1901, the largest percentage, viz. 12·66, were employed in agriculture, 11·39 in commerce, 7·89 in metals and machinery, 6·92 in textile fabrics, 6·77 in building and construction, and 5 per cent. in mines and quarries. In speaking of industrial evolution, Sir Thomas remarks that it cannot be denied that the monotony of modern factory life has dwarfed individuality, developed a kind of fatigue unknown to the working classes of two centuries ago, and a longing for excitement and recreation as a counterpoise to the depressing effects of work.

A rapid survey of the development of factory legislation shews what an immense improvement in the social condition of the workers has taken place during comparatively recent years. Interesting figures are given in illustration of the effects of dusty atmospheres on pulmonary consumption: thus, taking 100 as the mortality ratio of agriculturists,

potters give a ratio of 453; cutters, 407; filemakers, 373; glass makers, 335; workers in cotton mills, 244, etc. In illustration of the effect of fatigue and monotony in work, the author illustrates the fact by the Lancashire cotton-spinners, who, when they reach the age of sixty years, are quite unable to go on with the same work, and have to be given less well-paid employment.

That the conditions of work are not yet satisfactory is shown by the fact that the wages of women who make or carry bricks varies from 1d. to 2d. per ton, and that as a result of overwork much spinal deformity exists amongst brick workers.

We commend this little volume as one replete with interesting statistics and information invaluable to the social reformer as well as to the hygienist and medical practitioner.

Human Motives. By James Jackson Putnam, M.D. Pp. 175. London: William Heinemann. 1915. Price 5s. net.

This illuminative little book should be studied by any who still believe that man is a rational animal, in the sense of a being guided in his conduct chiefly by reason. To Dr. Putnam human activities are the outcome of motives of two kinds—the desires and instincts which belong to us by virtue of our evolutional history, and those which belong to us by virtue of our social relationships construed in the widest possible sense. These motives may be discussed from the philosophic standpoint or investigated by psycho-analytic methods. In either case one is led to a view of human progress as being equivalent to the discovery of new relationships between the inner world of one's own spirit and the world of empirical experience. The book will add to the reputation of the "Mind and Health Series" to which it belongs.

Sleep and Sleeplessness. By H. Addington Bruce. Pp. 219. London: William Heinemann. 1915. Price 5s. net.

This book, like Dr. Coriat's, belongs to the "Mind and Health Series." The author discusses the meaning of sleep, the mind in sleep, dreams, disorders of sleep, the causes of sleeplessness, and the cure of insomnia without recourse to drugs. Sleeplessness, according to the author, depends, as a general rule, on a state of mind, and is to be cured by inducing in the patient a new and healthier mental state. The book may be recommended as a summary of recent discussions and investigations.

Incompatibility in Prescriptions, and how to Avoid it. By THOMAS STEVENSON, Ph.C., F.R.S.E. Pp. 32. Edinburgh: *The Prescriber* Offices. 1915. Price 1s. net.

INCOMPATIBILITY is always a bugbear to the student, and often raises questions of difficulty in prescribing. This short pamphlet, reprinted from our contemporary *The Prescriber*, may be cordially recommended as a thoroughly practical and comprehensive guide, and a general knowledge of the principles so fully set forth ought to prevent mistakes arising. The original paper has been amplified by additional sections on explosive mixtures. It will be news to many, for instance, that our familiar family friends the hypophosphites are especially to be warned against in this connection, or that such an apparently harmless prescription as R Potass. chlorat. ʒiii., Tinct. ferri perchlor. ʒii., Glycerin. ʒii., is very liable to explosion. *Verb. sap.*: in these days of Zepps. one cannot be too careful.

NEW EDITIONS.

Operative Midwifery. By J. M. Munro Kerr, M.D., C.M.(Glasgow), Professor of Obstetrics and Gynecology, Glasgow University (Muirhead Chair). Third Edition. Pp. xvi. + 725. With 308 Illustrations. London: Baillière, Tindall & Cox. 1916. Price 25s. net.

One is not surprised to find a third edition of Professor Munro Kerr's well known and highly esteemed text book on *Operative Midwifery* on the table; but it says much for both author and publisher that the work has appeared in the form and with the rapidity that it has done in these strenuous times. It is a curious and a significant fact that war has rather turned attention to, than led it away from, midwifery; but it is easily to be explained, for if the present is to be the last of wars, then there will be tremendous gaps in manhood to be filled up, whilst if it is to be only the first of a series (surely an unthinkable thing), then recruits for the armies of the future must be found. In either case the preservation of infant life, and the bringing of as many infants as possible to the birth, demand the skill and call for the attention of the obstetrician. Professor Munro Kerr's volume really needs no lengthened review; it has supplied a felt need and filled a niche in medical literature satisfactorily, and it has received approbation and applause. Further, it is no mere assemblage of the views of others on the vexed questions of operative midwifery, but carries in every part the distinct impress of the author's own opinion, and enjoys the advantage of his wide experience. As might be expected, the chapter on Cæsarean section is excellent, although one misses any reference to Casalis's plan of delivery by hysterectomy, in which the womb is taken unopened from the abdomen and the child removed immediately thereafter—a plan which offers certain advantages in handled and presumably infected cases. The illustrations of Cæsarean section are most helpful in understanding the technic employed by the author. A little surprise will doubtless be expressed by some who read Professor Munro Kerr's comparatively mild criticism of the metallic dilators of the cervix, and his hopeful, if not optimistic, forecast of the probable results of cervical stretching by their means. These, however, are just the matters on which differences of opinion are expected to exist, and it is in their treatment that the individuality of the work is centred and finds expression.

The Principles and Practice of Obstetrics. By Joseph de Lee, M.D., Professor of Obstetrics, North Western University, Medical School, Chicago. Second Edition. Pp. 1087. With 938 Illustrations. Philadelphia and London: W. B. Saunders Co. 1915. Price 35s. net.

The fact that only two years have elapsed since Dr. Joseph de Lee presented the first edition of his *Principles and Practice of Obstetrics* to the profession is sufficient testimony of the excellent reception which has been accorded to it both in this country and in America.

The publishers, notwithstanding the introduction of much new matter, have succeeded by using thinner paper in preventing the volume, which was already of considerable size, from assuming clumsy proportions.

The enlarged chapters on such subjects as the Abderhalden reaction of pregnancy, extraperitoneal Cæsarean section, and "twilight sleep," are welcome additions. The article on "twilight sleep" is of particular interest at the present time, since the profession both in America and abroad have recently determined—so to speak—to give scopolamine morphine amnesia one more chance, after having practically abandoned it for several years. The author expresses the opinion that it can only be properly carried out in a maternity hospital, and that "its generalised re-employment will result in a repetition of the fœtal and maternal mortalities and morbidity of twelve years ago."

In the article on eclampsia the author states that in his experience the use of veratrone has not altered his results either way, and he now seldom uses it, and then only in the puerperium. We read with surprise and regret that this is his conclusion, because, since Dr. Haultain introduced the use of veratrone and placed his results before the Edinburgh Medical School, and subsequently before the profession at large, we have used it whenever opportunity arose, and have been able to corroborate all that Dr. Haultain claimed for it, never having seen it fail to stop the convulsions. The results have been so striking that we hope Dr. de Lee will not reserve veratrone for the puerperium, but will give it an extended trial in the eclampsia of pregnancy and labour.

Many new drawings have been added, and the Touch pictures are quite a feature of the illustrations. In the operative section the series of drawings illustrating Porro Cæsarean section are beautifully executed, but as this operation is so rarely performed as compared with the conservative one, we think the latter operation might in the next edition have one or two illustrations to itself.

The author himself hints that he may be rather dogmatic, but with that we have no fault to find, coming as it does from a

teacher of such wide experience, and in command of such a wealth of material.

The second edition may be expected to gain even a wider popularity than the first.

Transactions of the American Gynecological Society. Vol. XL., for the Year 1915. Pp. lv. + 545. Philadelphia: W. J. Dornan, printer. 1915.

THE annual volume of the *American Gynecological Society's Transactions* contains a wealth of interesting reading, some of it obstetric, some of it gynecological, and some of it rather foreign to both obstetrics and gynecology. But, indeed, medicine is not divided into water-tight compartments, and never should be; and although pyelitis is clearly a medical subject, its association with pregnancy brings it into obstetrics, whilst renal and ureteral calculus force their way in on account of the possibility of errors in diagnosis. Schumann writes in a comprehensive manner on hydrops universalis fetus, Martin and Chalfant deal with ovarian transplantation, and Sproat Heaney has some novel suggestions to make regarding nitrous oxide in labour; but all the articles are worth reading, and the Society has good reason to be satisfied with its annual output of work.

Diseases of the Nose and Throat, Comprising Affections of the Trachea and Œsophagus: A Text-book for Students and Practitioners. By Sir ST. CLAIR THOMSON, M.D., F.R.C.P. (Lond.), F.R.C.S. (Eng.). Second Edition. London: Cassell & Co., Ltd. 1916. Price 25s. net.

WE welcome a second edition of Sir St. Clair Thomson's text-book on *Diseases of the Nose and Throat*, a standard work upon the subject. The author in his preface introduces a quotation from Michelet—"*Un livre est toujours un moyen de faire un meilleur livre*"—and with this sentiment we agree, recognising that the author has improved upon his first publication, which appeared four years ago.

Our knowledge of the diseases of the upper air passages and our methods of dealing with them are always progressing, and the writer of a text-book must keep abreast of such progress. The previous volume has been carefully revised and new matter introduced where that has been found necessary. It is to the latter that we naturally turn in reviewing a second edition.

It is characteristic of this specialty that it is constantly extending its boundaries: the trachea, the bronchi, the œsophagus, and even the stomach have been brought under the eye of the trained laryngologist.

Now, the ophthalmologist is being relieved of his *bête noire*, the chronic dacryocystitis with stenosis of the lachrymal duct, because the nasal surgeon can drain the sac by establishing a permanent opening in the nasal cavity. The nasal route to the pituitary body is regarded by many as the way *par excellence* for dealing with tumours of that region, the sphenoidal sinus and the sella being reached after an extensive submucous resection of the nasal septum has been carried out. Each of these more recent developments is dealt with by the author in his special section upon operations. He tells us in his preface that he has entirely re-written the chapter upon the removal of the tonsils, and in it he gives a very fair account of the arguments for and against partial or complete removal of these structures. He decides, like the majority of laryngologists in this country, in favour of the latter method of treatment.

We feel sure that this volume, like its predecessor, will meet with the success which it fully deserves.

The Treatment of Fractures, with Notes upon a few Common Dislocations. By C. L. Scudder. Eighth Edition. Pp. 734. With 1057 Illustrations. Philadelphia and London: W. B. Saunders Company. 1915. Price 25s. net.

It can safely be stated that the eighth edition of this already well-known book will be received as a welcome friend by the medical man —whether he be in civil practice or on military duty, for undoubtedly the author has succeeded in giving a most comprehensive and up-to-date description of the immediate and later treatment of this important, and often extremely troublesome, branch of surgical practice.

The style is easy, and the illustrations, which include photographs of deformities following fracture, skiagrams, and X-ray tracings, are numerous and of a very high order. These points, and the fact that there is a very complete index, make the obtaining of information a matter of simplicity.

The earlier chapters are devoted to the treatment of individual fractures, while the last few chapters discuss special factors in the causation, diagnosis, and treatment of fractures in general.

In the latter group, the chapter on the treatment of gunshot fractures of bone deserves special mention. At the present time, the interest in this class of injury must be world-wide. In a large proportion of cases the treatment cannot be conducted along recognised lines, devolving upon the medical attendant a more constant supervision, and demanding from him a greater resourcefulness and initiative. The author, however, has met this contingency by giving in Chapter XXI. instructions in minute detail in the employment of the plaster of Paris bandage as a method of treating these open fractures.

An excellent account of the Rontgen rays and its relation to fractures is given in Chapter XX.

The last chapter deals with the more common dislocations, and should prove especially helpful to the busy practitioner.

Altogether, it is an admirable book, and worthy of the highest praise.

Emergency Surgery. By JOHN W. SLUSS, A.M., M.D. Third Edition. Pp. 832. With 682 Illustrations. London: William Heinemann. 1916. Price 17s. 6d. net.

THIS book is designed specially to assist the practitioner in the treatment of surgical emergencies. The author's descriptions are easily followed, and are written from a practical point of view. It must be admitted that most of the emergencies which one can think of are discussed in detail. One misses, however, a description of the operative treatment of perforated duodenal and gastric ulcers. A practitioner must certainly be prepared to operate in such cases, if special surgical skill is not available

In the present edition the subjects show evidence of careful revision. The chapter on military surgery has been entirely rewritten and embodies the results of the experience of surgeons in the present war. The large number of illustrations, derived from numerous sources, increase the value of the work.

A Manual of Surgical Anæsthesia. By H. BELLAMY GARDNER, M.R.C.S., L.R.C.P. Second Edition. Pp. xii. + 220. With 44 Illustrations. London: Baillière, Tindall & Cox. 1916. Price 7s. 6d. net.

THIS is a concise and well-written handbook of anæsthetics intended for the use of the student and general practitioner. The descriptions of methods of administration are admirably clear and are expressed in the dogmatic manner which is desirable in such matters. The author avoids the danger of expending too much space in explaining the mechanical details of the apparatus employed, these being, as he mentions, much more readily understood by demonstration.

The sections on spinal analgesia and the intratracheal insufflation of ether are very brief, and the merits or risks of the two procedures are not discussed in any detail. The section on the treatment of emergencies is also somewhat curtailed, but, as the author remarks, the greater part of the book is intended to indicate methods by which such complications will be avoided. On the whole, this is a book which can be recommended as a good practical guide to the administration of anæsthetics.

Textbook of Operative Dentistry. By Various Authors. Edited by C. N. Johnson, M.A., L.D.S. Third Edition. Pp. v. + 891. With 805 Illustrations. London: William Heinemann. 1915. Price 25s. net.

Seventeen different authors have contributed to this volume of operative dentistry, and as each writer has dealt with an aspect of the subject on which he is an authority, the work is of exceptional value alike to dental students and practitioners. The new edition presents to the reader the most advanced views on the subject. Its value is enhanced by two additional chapters on the treatment of impacted lower third molars, and on the application of the Röntgen rays to dentistry. The illustrations, including the radiograms, are appropriate and excellent.

Essentials of Medical Electricity. By E. Reginald Morton, M.D., F.R.C.S. Third Edition, revised and rewritten by Elkin P. Cumberbatch, M.A., M.B. Pp. xiv. + 303. With 83 Illustrations. London: Henry Kimpton. 1916. Price 6s. net.

The present edition of this book is a remarkable case of condensation, for in it the author has achieved the difficulty of producing a useful survey of medical electricity in a small space. Apart from a large number of good and helpful illustrations, the book is written in a style which is at once clear and concise. Particularly interesting are the chapters dealing with ionic medication, and the maladies for which electrical treatment may be applied. The book as a whole will be found instructive, for its pages focus much valuable information in a subject of absorbing interest.

NOTES ON BOOKS.

SINCE 120,000 copies of Mrs. Langton Hewer's *Our Baby for Mothers and Nurses* (John Wright & Sons, Ltd., Bristol, 1916, 1s. 6d. net.) have been sold since 1891, no criticism in one sense or another is likely to affect the popularity or position of this fifteenth edition. We cannot, however, recommend it as a satisfactory guide. It attempts far too much, and a great deal of the more purely medical advice given, though no doubt in harmony with the teaching of 1891, is becoming out of date. New wine has been poured too often into the old bottle, and practically all that is said about artificial feeding should be rewritten, or else very greatly condensed. Mrs. Hewer is much more inclined to dogmatise on matters of detail than most of those who have followed recent progress in artificial feeding, and teaching such as she offers is calculated to lead to false impressions being formed by mothers who have no other guidance, and no experience to enable them to qualify her words.

The twenty-seventh volume of the *Transactions of the American Pediatric Society* affords ample proof, if any were needed, of the valuable work done by the American School of paediatrists. The thirty-four articles in this volume cover a wide field, and the contributors include many of the best known names in America. As usual, work in connection with the metabolism of infancy and with questions of diet bulks largely, but no outstanding advance in this department falls to be mentioned. One of the most interesting papers read, which really opens up a comparatively new field of observation, is that by Dr. Oscar Schloss, on allergy to common foods. The point of interest raised is, whether it may not be possible to isolate the peccant article of food which is, according to the current hypothesis, responsible for asthma, eczema, and possibly some form of diarrhœa, by means of cuti-reactions. For example, in a case of eczema, can one by inoculating, "von Pirquet" fashion, the various food elements the infant is taking, and by eliminating from the diet those to which there is an allergic reaction, go any length towards curing the eczema? Can one, dealing with a child who is known to have been in early infancy intolerant of cow's milk, by the cuti-reaction determine when this susceptibility is lost? Dr. Schloss' experiments along these lines are at all events suggestive of future possibilities. There are also in this volume a number of other papers which will repay persual.

BOOKS RECEIVED.

HOFMANN, FRIEDRICH L. The Mortality of Cancer throughout the World. (*The Prudential Press*)

THE Declining Birth-Rate: Its Causes and Effects . . . . . (*Chapman & Hall*) 10s. 6d.

VARLEY, KITSON. Food Culture . . . . . (*Hodder & Stoughton*) 2s. 6d.

FORTHCOMING PUBLICATIONS.

The following New Books are announced :—

MESSRS. J. & A. CHURCHILL.

Gynecology for Students and Practitioners, by Dr. T. W. Eden and Dr. C. Lockyer.

The Story of a Red Cross Unit in Serbia, by Mr. and Mrs. Berry, Mr. W. Lyon Blease, and other members of the unit.

EDINBURGH MEDICAL JOURNAL.

EDITORIAL NOTES.

Turner Memorial Number of "Student."

The editorial staff of the *Student* are to be congratulated on the special number of the magazine devoted to the memory of the late Principal of the University. The task of compiling such a tribute as would be worthy of Sir William Turner and his work for the University was no light one in these days of strain and stress, but the editor has succeeded in accomplishing it. Appreciations of Sir William appear from the Chancellor, Mr. A. J. Balfour; the Member for the University, Sir R. B. Finlay, who writes as a former pupil; from the Secretary of the Senatus, Sir Ludovic Grant; and from the President of the General Medical Council, Sir Donald Macalister, who deals with Turner as educationalist as well as anatomist. Emeritus Professor J. G. M'Kendrick contributes most interesting recollections of his life long friend, and the Principal of Aberdeen University, Sir George Adam Smith, pays a warm tribute of honour and affection to his memory. Dr. John Horne—Turner's successor in the Chair of the Royal Society of Edinburgh—records the valuable work his predecessor did the Society as Councillor, Secretary, and President. Turner's share in carrying through the building of the New University at Teviot Place, and its completion by the erection of the M'Ewan Hall, is fully related by Mr. George Somerville, formerly Clerk to the M'Ewan Hall Trustees. Lord Kingsburgh contributes a characteristically kind and genial appreciation; and Dr. John Kelman recalls the constant sympathy he received from Sir William in the work he carried out for so many years at the Operetta House. To scores of old Edinburgh men scattered over the wide world this article will recall those hours in their University course which did most to mould their character and to strengthen their faith. Mr. George W. Dunlop writes of Turner from the student's point of view with insight and sympathy, and the volume ends with a report of the ceremony of presenting the Guthrie portrait of Turner to the University in 1913.

A series of characteristic portraits of Sir William are beautifully reproduced, as well as portraits of most of the contributors to the

Memorial. Old Edinburgh men will appreciate also the excellent series of views of the M'Ewan Hall and its mural decorations. The cover, representing a kilted soldier placing a tributary wreath, is peculiarly appropriate at this time, when so many of our students are on active service, and is from the pen of Joseph Simpson, a member of the Royal Society of British Artists and a frequent contributor of sketches to the *Student*.

CASUALTIES.

KILLED in action, Lieutenant DOUGLAS RODGER, R.A.M.C., on 1st July, aged 33.

Lieutenant Rodger was educated at Trent College, at Edinburgh, and at Manchester, where he graduated as M.B. and Ch.B. of the Victoria University in 1907, also taking the F.R.C.S.(Edin.) in 1912. He took a temporary commission in the R.A.M.C. last September, and was working in an advanced dressing station when killed.

KILLED in action, Captain THOMAS ERROL GUTHRIE, R.A.M.C., on 4th July.

Captain Guthrie was educated at Edinburgh University, where he graduated as M.B. and Ch.B. in 1909, joining the Special Reserve of the R.A.M.C. as a lieutenant on 17th December 1914. Prior to the war he was in practice at Fielding, New Zealand.

KILLED in action, Captain E. W. LAWRENCE, on 10th July, aged 28.

Captain Lawrence was educated at Cardiff and Edinburgh, taking the degrees of M.B., Ch.B.(Edin.), in 1910. He was medical officer to one of the service battalions of the Welsh Fusiliers.

KILLED in action, Captain DAVID WALLACE SMITH, R.A.M.C., on 15th July.

Captain Smith was educated at Glasgow University, where he graduated as M.B. and C.M. in 1901, and was in practice at Brixton. He joined the R.A.M.C. in August 1914, and was attached to the Manchester Regiment.

The following students of medicine have appeared in recent casualty lists:—

DIED of wounds received in France on 2nd July, Second-Lieutenant WILLIAM ADRIAN DAVIDSON, Gordon Highlanders, aged 21.

Lieutenant Davidson was engaged in medical studies at the University of Aberdeen prior to obtaining a commission in the Gordon Highlanders.

KILLED in action, Lieutenant LACHLAN MACKINNON, Cameron Highlanders, aged 25.

Lieutenant MacKinnon, previous to obtaining his commission, was a medical student at Edinburgh University.

The Summer Graduation in Medicine.

THE Summer Graduation Ceremonial was held in the M'Ewan Hall on Tuesday, 14th July, when the new Principal, Sir Alfred Ewing, presided and, after conferring the degrees, delivered the address to the Graduates.

The Degree of Doctor of Medicine was conferred on the following: Allan Stone Boyd, Scotland, M.B., C.M., 1893, "The Treatment of Asthma by Cauterisation of the Nasal Septum" (*in absentia*). Robert Govan (M.A.), Scotland, M.B., Ch.B., 1910, "On External Tuberculosis, that is to say, the Manifestations of Tuberculosis which present themselves Superficially on the Human Body." William David Stoney Johnston, New Zealand, M.B., Ch.B., 1907, "On Contracted Pelvis, with Special Reference to the Treatment of Various Degrees of Contraction, and the Employment of the Operation of Pubiotomy, with Illustrative Cases" (*in absentiâ*). John Lindsay, Scotland, M.B., Ch.B., 1905, "Industrial Lead Poisoning: Its Prevention and Treatment." Gabriel Julian Luyt (B.A., Cape), South Africa, M.B., Ch.B. (with 2nd class Hons., 1907, "The Pituitary Body and its Extracts in Pregnancy and Labour" (*in absentia*). Robert Mitchell Mackay, New Zealand, M.B., Ch.B., 1914, "Some Investigations on the Detection of Occult Blood in Stools, with a Simplified Technique for the Application of the Benzidin Test." Alexander William Mather (Lieut., R.A.M.C.), Scotland, M.B., Ch.B., 1912, "Prolapsus Uteri, with Results of One Hundred Cases Treated by Vaginal Plastic Operations" (*in absentia*). Adam Niven Robertson, Scotland, M.B., Ch.B., 1907, "The Treatment of Tuberculosis by means of Spengler's Immune Bodies (I. K. Therapy)" (*Highly Commended for Thesis*). Chung Chung Wang, China, M.B., Ch.B., 1911, "Studies on the Early Stages of Development of the Blood Vessels and of the Heart in Ferret Embryos, with a Comparison between the Conditions Observed in the Ferret and those Noted in other Mammals of relatively the same Stage of Development (*Gold Medal for Thesis*). Ching Yik Wang, China, M.B., Ch.B. (with 2nd class Hons.), 1910, "A Study in some Phases of Tuberculosis, with Special Reference to the Incidence of Bovine Infection and the Question of Latency and Prevalence of the Disease" (*Gold Medal for Thesis*). Walter Quarry Wood, Scotland, M.B., Ch.B., 1911, "The Australian Tibia" (*Gold Medal for Thesis*).

The Degree of Doctor of Science was conferred on James Walker Dawson, M.D., "The Histology of Disseminated Sclerosis."

The Degrees of Bachelor of Medicine and Bachelor of Surgery were conferred on Charles Bevan Carew Anderson, Ireland (Lieut., R.A.M.C. (S.R.)); Gerald Hugh Barry, England (Lieut., 13th Lancs. Fusiliers); John Bennet, Scotland (2nd class Hons.); Roderic Duncan Cameron, Scotland (Lieut., R.A.M.C. (S.R.), Capt. Arty., O.T.C.), Jerry Ewing Chow, British Guiana (*in absentia*); Ando Huan Chu, China; Demetrius Colombos, Malta; Walter James Currie Craig, Scotland (Lieut., R.A.M.C. (S.R.)); Thomas Crisp, Australia; Alfred Lucian Vincent Davin, Morocco; Donald Gordon Dull, Scotland; John James Palmanno Edmond, Scotland (Lieut., R.A.M.C. (S.R.)) (1st class Hons.); Archibald M'Laren Ferrie, Scotland; Malcolm Foster, Scotland; Arthur Keith Gibson, Scotland; William Niven Greer, Ireland (2nd class Hons.); François Lambert Hauptfleisch, South Africa; Carl Sebastrom van Heerden, South Africa; Norman Keane Henderson, Scotland (Surg. Prob., R.N.V.R.);

Wilhelmina Weir Hendry, Scotland; Siddha Nath Kaul, India; Marjorie Isobel Stuart McGregor, Scotland; Ian MacKenzie, Scotland; John Alexander Mackenzie, Scotland; Isobel Mary MacLullich, Scotland; John Oloff Marais B.A. Cape, South Africa; Jacob de Vos Meiring, South Africa; Robert Watson Hamilton Miller, Scotland; Everard Arnold Mills, New Zealand; Colin Milne, England; James Milne, Scotland; Hamilton Stephen Moore, Scotland; John Lewis Owen, Australia (1st class Hons.); Arthur McMaster Paterson, Scotland (Lieut., R.A.M.C. (S.R.), Capt. Att., O.T.C.); Clifford Pearce Penberthy, South Africa; Adam Prentice, Scotland; John Campbell Preston, Ireland; William Richards, Wales; Benjamin James Ryrie, Scotland 1st class Hons.; Eng Siang Seah, China; Ernest Oscar Adolphus Singer England; James Maclure Smellie, England; Erik Daniel Soderstrom, Sweden; Murdo Stewart, Scotland; Robert Bell Stewart, Scotland (Capt., 12th Batt. R.S.); Douglas Garnett Stoute, British Guiana; Mukundrai Dolatrai Thakore B.Sc., India; Daya Ram Thapar, India; William Goldie Thomson, South Africa; Jacob Isaac de Villiers, South Africa; William Waddell, South Africa; Richard Robert Stevenson Weatherson, Scotland; Gladys Richarda May Violet Williamson, Ireland; Robert Maclaren Wishart, Scotland (Lieut., R.A.M.C.); Charles William Ng Yow, China.

The following awards were made:—

Thesis Gold Medals to Chung Ching Wang, M.D.; Chung Yik Wang, M.D.; Walter Quarry Wood, M.D. *The Ettles Scholarship* to Benjamin James Ryrie, M.B., Ch.B. *The Beaney Prize in Anatomy and Surgery* to Alfred Vincent Dill, M.B., Ch.B. (Lieut., R.A.M.C.). *The Mount Scholarship in the Practice of Physic* to Andrew James Caird, M.A., B.Sc., M.B., Ch.B. (Lieut., R.A.M.C.). *The Conan Doyle Prize* to Cecil Thomas Ingram Clarke, M.B., Ch.B. *The Buchanan Scholarship in Gynæcology* to Benjamin James Ryrie, M.B., Ch.B. *The James Scott Scholarship in Midwifery* to John Bennet, M.B., Ch.B. *The Dorothy Gilfillan Memorial Prize* to Gladys Ward, M.B., Ch.B. *The Pattison Prize in Clinical Surgery* to John Crawford Burns. *The Cunningham Memorial Medal and Prize in Anatomy* to Morris Goldberg. *The Whiteside Bruce Bursary* to John Rough McDonald. *The Crum Brown Medal in Chemistry* to George F. Davidson.

The following candidates have completed the undermentioned University Professional Examinations:—

First Professional.—Isobella M. M. Aitken, Harry Alexander, Sydney E. Ammon, Henry V. Anderson, Charles G. Auld, Charles J. Banks, William Boyd, Grace S. Calver, Helen Campbell, Clarence H. F. de Chabus, Nicholas S. Craig, Douglas R. Cramb, John Davidson, Edward D. Dingle (*with distinction*), Katrine Dunn, Raymond W. Dupre, William Eadie (M.A.), Grace W. W. Eason, Isaac Eban (M.A., B.Sc.), Basil A. G. A. Edelston, Joseph H. Fairweather (*with distinction*), Hugh M. Forbes, George D. Fraser, Dorothea M. Gall, Robert M. Galloway, Beatrice C. Galsworthy, George A. V. Gardiner, Marjorie Geen, Mary L. Gilchrist, Isabella A. Gillespie, James A. Gillison, Elspeth L. Gilmour, John B. Given, Jessie Gordon, Janet Grant (*with distinction*), Winsome D. Grantham, William L. Grassick (M.A.), Alice J. Gray (M.A.), John D. Grierson, John E. Haine (*with distinction*), Arthur J. C. Hamilton, Christina M. Hawick (B.Sc.), Harold H. Holden, Robert G. Inkster, Roderick A. N. James, Andrew D. Kincaid, Samuel Lipetz, Ian H McClure (*with distinction*), Angus Macdonald, Flora MacDonald, John R

Macdonald (*with distinction*), William R. K. MacGillivray, Agnes M. Macgown, Henry Mackay (M.A.), Florence E. McKenzie, Henry D. McKinnon, A. Sheila MacLeod, Mona Macnaughton (M.A.), Jean S. B. McNeil, Christian B. F. Millar, Quazi M. Musa, Jessie S. Paton, William P. Perrie, Helena J. Robertson, Charles A. Robson, Eileen S. P. Rowney, Annie M. Roxburgh, Helen M. Russell, Veronica G. Salvesen, Gordon S. Scott, Dorothy M. Somerville, Thomas Spruat, Marguerite Stewart, E. Muriel Stokes, Sydney S. Sumner, John B. Taylor, Lewis Teitelman, Elizabeth S. Thomson, Mabel Thomson, David W. C. Traigh, Arthur K. Towers, David J. Whitton, Anna M. Williams, Christine V. D. Willway.

Second Professional.—Adam Armit, William L. Balfour, Alice Bloomfield, Harold C. Brayshaw, Donovan Brink, Donald A. Carlman, Charles G. Cates, Norman H. Clarke, James M. Cole (*with distinction*), Montague Danziger, Andrew W. Davison (*with distinction*), Norman W. Dott, Millicent Fox (B.A.), Dora W. Gerrard, Morris Goldberg (*with distinction*), George H. Gunn, Arthur W. P. Haine, Marjorie Harris, James A. Hennessy, John P. Hope, Lawrence J. Howie, Ralph A. S. Hoyte, William Isbister, James L. Lloyd Jones, William Kantor, Ba Kin (B.A.), Albert C. Lornie, Thomas W. Lowden, Sylvester H. Mearns, P. Sankara Menon, Harvey Nichol (M.A.), Pysika P. Rao, D. Gordon Robertson, Janet C. Rugg, Charles E. Scott, Muriel Sellers, S. Solomon Silva, Bertie S. Simpson, George A. Sinclair, Sardar C. Soni, Daniel A. Stegman, Petrus C. Uys, Ardeshir J. Vakil, Vera C. Veitch, Alfred G. N. Weatherhead, Wyatt L. Zeeman.

Third Professional.—George G. Allan, George S. Bainbridge, Irving A. Beckles, Cecil Blake, Ruby T. Carr, Edward Chonglop, Albert C. Y. Chow, Edward D. D. Dickson, Isobel M. Don, Johannes K. van O. Dunnay, Duncan Fergusson, Richard N. Gibson, Sukumar D. Gupta (*with distinction*), C. Edith L. Hole, Donald C. Lamont, Robert K. S. Lim, Robert B. MacGregor, Albert S. M'Kern (B.A.), Alexander R. C. M'Kerrow, James M'Nabb (M.A., B.Sc.), Robert C. B. Macrae (*with distinction*), Moray Melvin, J. Kenneth Mitchell, Joyce C. B. Mitchell, Alexander J. Muirhead, Samuel D. Nurse, Henry J. Parish, Hugh S. Percival, Israel Platzky, Frederick W. Poole, Ernest G. Pratt, Joseph Rauch, Charles B. B. Reid, Horatio B. Renton, Gertrude M. V. Richardson, Max Sash, Charles Simpson, Frank W. Simson, David W. Sinclair, G. M. Shaw Smith, Janet S. Smith, John H. R. Smith, John C. Truter, Maung S. Tun (B.A.), Jakob R. de Villiers, Ulric G. Williams.

ROYAL COLLEGE OF SURGEONS OF EDINBURGH.

The following have been admitted Fellows of the College after examination:—Ivan Wanless Dickson, M.B.(Toronto Univ.); George Hamilton Ross Hamilton, M.B.(Toronto Univ.); William Howard Thomas, M.B., Bac. Surg. New Zealand Univ.; Henry Richard Hopkinson, M.B., M.S.(Sydney Univ.); Wesley John Jenner, L.R.C.S.E.; Bireswar Mitra, L.M.S.(Calcutta Univ.); Charles William Sharp, L.R.C.S.E.

TOXI-INFECTION OF THE CENTRAL NERVOUS SYSTEM: A CLINICAL AND EXPERIMENTAL INVESTIGATION.*

By DAVID ORR, M.D., and Major R. G. ROWS, R.A.M.C.

The obscurity which surrounds the genesis of almost all inflammatory lesions in the central nervous system, and of those which are degenerative except where a focal lesion exists, will be admitted. The questions of the causation of the lesion, the point of origin of the morbid change and its propagation, are constantly recurring in regard to cases of meningitis, myelitis, tabes dorsalis, dementia paralytica, and the non-systemic scleroses, and the theories advanced have often been based on assumptions devoid of proof which have tended rather to divert the investigator from, than to lead him on to, the right path. Nor should one be astonished that this is so, for the intricate anatomy of the nervous system, its complexity of function, and its comparative inaccessibility to experimental methods have taxed the ingenuity of the investigator to the utmost. Hence the theories regarding the genesis of many morbid phenomena have been based upon deductions built up on the examination of tissue in which the chronicity of the changes obscured the original starting-point of the retrograde or inflammatory process. This applies particularly to subacute and chronic lesions, systemic and non-systemic, affecting the columns of the spinal cord.

For years it has been apparent that continued examination of these chronic lesions while increasing our knowledge in detail yet failed to widen it in regard to etiology, and though toxic influence naturally received due recognition, its source and mechanism of action remained unexplained. It seemed obvious, therefore, that investigation ought to be directed towards elucidating the mechanism of production of those lesions, and the first step naturally involved a study of all possible paths of infection and intoxication. It is with one of these—infection *via* the lymphatic system of peripheral nerves—that the first portion of this paper deals.

In all probability the controversy surrounding the genesis of

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the lesion in tabes dorsalis and the recognition of dementia paralytica as an inflammatory disease were most important factors in directing attention to the lymphatic system of the cerebro-spinal axis, and an important step forwards was taken with the demonstration of a continuity of the lymph-stream in peripheral nerves with that of the spinal cord. The injection into nerves of organisms and coloured substances showed that the lymph stream was an ascending one towards the cord and that the main current lay at the periphery of the nerve-bundles immediately under the fibrous sheath. It must be borne in mind, however, that this statement regarding the main current of lymph in nerves was based on experiments in which organisms had been injected into the nerve substance. Where infection occurs from without it will be shown that diffusion of organisms and toxins can take place along the outer surface of nerves and give rise to an ascending epi- and peri-neuritis.

Infection along peripheral nerves had been deduced for many years in regard to tetanus and rabies, and the recognition of a wider application of this principle suggested a new line of research along which an explanation of certain other pathological phenomena might be sought.

Our research commenced in 1903, when we had the opportunity of examining the nerve tissues in a case of left brachial neuritis of twelve days' duration. The exciting agent in this instance was the *staphylococcus pyogenes aureus*. We found the brachial plexus bathed in pus which had burrowed among the cervical muscles as well, and surrounded the root ganglia of the cervical cord. The microscopic examination of the tissues proved of great interest. The loose areolar tissue round the spinal root ganglia of the left side was greatly inflamed, the veins were thrombosed and contained many cocci. Those not thrombosed were greatly congested; the arterioles, on the other hand, showed no morbid change. In the ganglion capsule there were hæmorrhages and cocci, more evident in the outer layers, and amongst the nerve-cells dilated vessels and capillaries containing micro-organisms. Similar changes affected the perineurium of the brachial plexus, but to a less degree. No cocci were found in the dura of the cervical cord or loose areolar tissue covering it. One small group only was observed in the pia-arachnoid covering the posterior columns. There were no inflammatory phenomena in either membrane. The 6th and 8th cervical root ganglia of the right side were examined. There were no thrombi in the capsular areolar tissue.

but scattered haemorrhages in which were a few cocci. No cocci were found in the ganglion substance.

The extramedullary portion of the anterior and posterior cervical roots was examined for degeneration by Marchi's method and none was found. In the spinal cord, however, the same method demonstrated an acute degeneration whose distribution was confined to the cervical region. The seventh, sixth, and fifth segments showed much the greatest degree of degeneration. There was marked degeneration of the intramedullary portion of the left posterior root, which, commencing at the point where the fibres lose their neurilemma sheath, affected the middle area of the root entry zone. On the right side the degeneration affected the same area, but was less marked. There were degenerated fibres on each side of the postero-median septum. In the lateral columns of the cord on either side of the septa derived from the pia-arachnoid there was a considerable degree of myelin degeneration; and here and there in the posterior and lateral regions there were degenerated myelin droplets at the cord margin. A considerable degree of degeneration was observed in connection with the intramedullary portion of the anterior roots in their whole course from the cord periphery to the grey matter. There was degeneration of the collaterals running from the left root entry zone to the anterior cornual cells, and also of the anterior commissure. All the vessels of the cord and meninges were greatly congested.

The observations in this case were confirmed and amplified by examining the spinal cords from cases of bedsores, pelvic cellulitis, septic psoas abscess, chronic suppuration of the knee-joint, and transverse myelitis secondary to prostatic cancer with chronic cystitis.

All the above cases were studied by the Marchi method for myelin degeneration, and all showed involvement of the posterior columns in that region of the cord which corresponded to the nerve supply of the infective focus. At times also the anterior radicular fibres were affected. Another noteworthy point is that the degeneration of both anterior and posterior spinal roots always begins at the point where the fibres entering the cord lose their neurilemma sheath, that is in the intramedullary portion, and in the case of the anterior roots the degeneration in some instances has been traced from the cord margin into the grey matter, where it terminated among the cells. The effect of the toxins is not always exerted on the lateral portions of the cord. In the case

of brachial neuritis, however, we find the lateral diffusion very marked, a fact to be explained in all probability by the high activity of the infective focus and its close proximity to the spinal cord. The greater the degree of toxicity, therefore, in the lymph-stream, the more diffuse the cord lesion.

Evidence of Intrapiaqueous Infection of the Central Nervous System from Experiment: Degenerative Lesions.—When the examination of clinical material had shown that lesions in the central nervous system had a definite anatomical relationship with the nerve supply of peripheral infective areas, and that these lesions depended upon toxic diffusion along the perineural lymphatics, it seemed that to submit this view to experiment would be the most certain method of obtaining definite data.

Instead of injecting organisms or toxins into the nerves on which it was proposed to operate, we placed a celloidin capsule containing a broth culture of a micro-organism in contact with the nerve. This procedure was adopted at the suggestion of Professor Lorrain Smith, in whose laboratory at Manchester we conducted our experiments. In one series of experiments this was placed under the gluteal muscles alongside the sciatic nerve; in another series under the skin of the cheek. The animals experimented upon were rabbits and dogs, and the organisms used were *staphylococcus pyogenes aureus*, *bacillus pyocyaneus*, Gaertner's bacillus, *B. coli*, *B. botulinus*, and a culture of a diphtheroid bacillus obtained from a case of dementia paralytica. We commenced experimenting with all these bacilli, but later adopted a strain of *S. aureus* whose virulence had been raised. It was also found expedient to renew the capsule at intervals. Some of these capsules remained intact, others did not, and a variable amount of leakage occurred. The tissues from this series of experiments were prepared for examination of the degenerative changes in the medullated fibres of the nerves, cord, medulla, and pons. Marchi's osmic acid method was employed.

In those experiments in which the capsule was placed in contact with the sciatic nerve a degeneration of the exogenous system of fibres in the posterior columns of the cord was induced, which commenced where the fibres lose their neurilemmal sheath and involved the root entry zone and the collaterals passing into the grey horns.

When the capsule was placed under the skin of the cheek the intramedullary portion of the cranial nerves in the pons and medulla showed a variety of degenerative changes. For example,

in the 5th the degeneration was Wallerian in type; in the 3rd and 6th, well-marked "segmentary necrosis." The extramedullary portion of all the nerves was normal.

From the clinical cases and the results of experiments we arrive at the following conclusions:—(1) In spinal and cranial nerves there is an ascending lymph-stream to the central nervous system whose main current lies in the spaces of the perineural sheath. Toxins reach the spinal cord and brain by this route; and although they spread to some degree in the lymph spaces of the pia-arachnoid, and so may affect structures at a distance from the point of entrance, they pass for the most part in the main stream along the nerve roots into the substance of the central nervous system. (2) Outside the central axis the nerves are possibly protected by the vital action of their neurilemma sheath; most probably, however, it is the peripheral situation of the lymph current which is the deciding factor.

The Histological Evidence of Experimental Infection of the Central Nervous System by the Lymph System of Peripheral Nerves.—We now repeated the above experiments and treated the tissues in a manner suitable for the demonstration of the histological reaction.

In one series the capsule was placed against the sciatic nerve; in the other, the spinal column was denuded of muscle and the capsule laid close to the intervertebral foramina.

On examination all the signs of an ascending perineuritis were apparent, extending from the infective focus as far as the cord, and there the inflammatory reaction showed all the phenomena of a meningo-myelitis, which attained its maximum when the capsule placed close to the intervertebral foramina had burst, and the organisms escaped into the surrounding tissues.

The evidence from these experiments leaves no room for doubt that the lymph stream in nerves is an ascending one, and that toxins and organisms can be carried to the cord by that path. The reaction of the tissues to the toxin also shows that the lymph not only ascends in the spaces of the nerve sheaths, but diffuses in the fibrous septa between the nerve fasciculi and into the adventitia of the vessels. From the experiments, too, it is clear that when the toxin gains the spinal cord it is carried round the periphery in the meshes of the pia-arachnoid and along its prolongations into more central parts. It is exceedingly interesting to observe how the character of the inflammation undergoes progressive changes from the focus of greatest intensity onwards; and there is one

important fact to which we would draw attention, and that is, how plasma-cell formation becomes the most prominent indication of irritation when the irritant has been to a great extent neutralised by the reaction of the tissues close to the capsule.

The proliferation of the adventitial sheath of the veins and capillaries with plasma-cell formation shows that its lymph spaces communicate directly with the lymphatic system of the nerves and cord: and though, doubtless, the adventitial lymph system of the arterioles has a similar connection, yet the intense reaction around the veins and capillaries points to these as being the principal channel by which the lymph regains the general circulation. This opinion receives support from researches carried out by other methods, such as the injection of coloured fluids into the sub-arachnoid space; these find their way rapidly into the adventitia, and thence into the blood-stream. According to Lewandowsky, the veins, capillaries, and Pacchionian bodies are the channels by which the cerebro-spinal lymph is returned to the general circulation. The reason why the adventitia of the veins and capillaries of the nervous system in lymphogenous infection should show so much inflammatory change is therefore obvious.

The results of the above experiments show that infection of the lymph system of peripheral nerves is followed by an ascending perineuritis which spreads to the posterior root ganglia and along the spinal roots to the cord. The loose areolar tissue covering the perineurium, the ganglion capsule, and the dura mater shows the greatest degree of inflammation, and along with these fibrous structures forms a natural and efficient protection against infection to the underlying nervous elements.

At this stage of our experimental investigations we supplemented our researches by several clinical observations. We examined in collaboration with Dr. Stephenson of Whittingham Asylum, the central nervous system of 7 cases, viz.:—(1) Carcinoma of the tongue with suppuration of the floor of the mouth; (2) erysipelas of the face; (3) juvenile general paralysis with bed-sores over sacrum, right hip, and elbow; (4) tubercular nodules in pleura; (5) tubercular lumbar abscess, the patient dying of acute meningitis in 48 hours; (6) cancer of œsophagus; (7) empyema of right pleural cavity, with herpes zoster on the same side.

All these cases, except the last one, showed a diffuse meningo-myelitis of the cerebro-spinal axis, the result of toxi-infection of the peripheral nerves. The inflammatory phenomena in the cord

and its membranes vary in intensity from case to case according to the potency of the infective agent, and the spread of the inflammation is by the direct continuity so typical of lymphogenous infection. The cases show that any portion of the central nervous system may be attacked by organisms or toxins passing up the nerves from infective foci, and although the morphological changes may vary according to the potency of the exciting cause, the anatomical path of entrance and spread is a constant one.

We have already demonstrated how in experimental infection of the lymph-stream of nerves certain structures invariably show the greatest degree of reaction, and the above clinical cases confirm this. These structures are the loose areolar tissue covering the nerve sheath and the dura mater, and the adventitia of the veins and capillaries. In both the peripheral nerves and cerebro-spinal axis the degree of reaction diminishes from without inwards, and that level of the latter directly connected by nerves with the peripheral source of infection exhibits the highest degree of inflammation.

So far we have dealt only with lymphogenous infection of the cord and brain. We shall now contrast the lesions produced by this mechanism with those which occur when a haematogenous intoxication is induced.

We undertook a series of experiments in which the abdominal cavity was chosen as the site for infection. This was done for three reasons:—(1) The peritoneal cavity is most suitable for an experiment in which one wishes to avoid infection of the lymph system of spinal nerves; (2) to reproduce as closely as possible a gastro-intestinal intoxication, and observe the effects upon the spinal cord; (3) to ascertain in how far such toxi-infection affected the sympathetic ganglion chain. Celloidin capsules containing a broth culture of the *staphylococcus pyogenes aureus* were therefore placed in various regions of the abdomen, where they became attached to the mesentery, kidney, bladder, or lower border of the stomach, etc.

On examining the spinal cord from this series of experiments we found no evidence of lymphogenous invasion, either in the sheath of the spinal ganglia, the perineurium of the spinal roots, or in either the dura mater or pia-arachnoid. Within the cord, however, there were very definite changes, which we will summarise shortly:—(1) The most highly developed structures, the nerve-cells, suffer least of all; (2) there is primary degeneration of the myelin sheath round the cord margin and along the

postero-median septum; (3) the myelin degeneration is greatest in the upper part of the cord; (4) there is œdema of the cord; (5) there is active proliferation of the perivascular neuroglia; (6) the vessels are dilated, congested, are hyaline, and contain thrombi of the same nature. If these be now contrasted with the cord lesions in lymphogenous infection the difference is at once obvious. Lymphogenous infection is characterised by (1) the reaction of the cells of the fixed connective tissue; (2) the proliferation of the cells of the adventitial sheath of the veins and capillaries; (3) the appearance of numerous scavenger-cells when the myelin is disintegrated; (4) nerve-cell degeneration and neuronophagy. From the above, one must conclude that the lesions in hæmatogenous intoxication differ very widely from those found in lymphogenous infection, where the fixed tissues are actively proliferating and all the morbid phenomena are of an inflammatory type. The difference between the two might, therefore, be expressed by saying that in lymphogenous infection the inflammatory phenomena reach their maximum, in hæmatogenous intoxication they are reduced to a minimum, and we consider this a most important distinction in neuropathology.

The condition of hyaline thrombosis referred to above varies in degree and in appearance from case to case. Arteries, veins, and capillaries are affected, and the thrombotic change is accompanied by œdema, shown by wide dilatation of the perivascular and pericellular spaces.

In incomplete thrombosis the hyaline material lies along each side of the vessel as a thick band, the two sides being connected by trabeculæ, thus forming a network in the lumen of the vessel. Frequently, however, the whole lumen is occupied by a homogeneous hyaline mass, or by a structure in which many red corpuscles are clearly evident in various stages of hyaline degeneration.

The leucocytes also undergo hyaline degeneration. Their affinity for acid fuchsine is intensified, they lose their normal shape, become clumped into masses in which are numerous granules which stain deeply with hæmatoxylin and are at times arranged in a horse-shoe fashion. These granules we regard as remains of degenerate leucocyte nuclei. There are many hyaline threads in the vessel lumen to which the leucocytes frequently contribute, forming a hyaline syncytium. Purely fibrinous thrombi are not infrequently met with.

In giving a short account of these experimental hæmatogenous lesions, we feel that the term "hæmatogenous" requires qualifi-

tion. We do not consider that the term sufficiently explains the mechanism of production. It would seem as though there was another factor at work. If we look at the distribution of the myelin degeneration alone for a moment, round the cord margin where œdema is present, and on either side of the postero-median septum, the explanation of a "general intoxication" is hardly sufficient. Weaker resistance on the part of the fibres in those situations might be put forward as a predisposing cause, but this suggestion cannot be substantiated.

Lesions of practically the same distribution are found in cancer cachexia, pernicious anæmia, Addison's disease, etc. It is of importance to note that the grey matter of the cord so richly supplied by the anterior spinal artery may be normal; Goll's tract in the cervical enlargement is attacked in its middle third, leaving the long lumbo-sacral fibres so far away from their trophic centre untouched; between the lesions in the cervical, dorsal, and lumbar cord there are great differences not to be accounted for by a focal lesion. Such a distribution of degeneration cannot be explained by a general intoxication. It fails completely when we consider that the areas supplied by the pial vessels, the cord margin and postero-median septum, show degenerative change, while the regions supplied by the anterior spinal arteries escape. It is highly probable that there must be another factor besides intoxication which determines the localisation of the degenerations, and certain indications point to the sympathetic nervous system.

We have shown evidence of involvement of this mechanism in the inflammation round the sympathetic ganglia, the proliferative changes in the adventitia of their vessels, and in the chromatolysis of the nerve-cells; and when it is remembered that the cord lesion in abdominal cancer and Addison's disease—in both of which sympathetic reflex action must be considerably disturbed—have almost the same distribution as in our experiments, sympathetic influence cannot be excluded.

There is evidence in the vessels of the cord, too, of altered sympathetic action; in the dilatation, hæmorrhages with sclerosis, and in the hyaline thrombosis—all pointing to stasis. As to whether this stasis is due to toxic action upon the prevertebral chain, or is caused by the direct action upon the cord vessels of a toxin in the general circulation, it is difficult to say. This latter suggestion seems improbable, as judging from the relative integrity of the nerve-cells and the minimal reaction of the vessel endothelium within the cord, the toxicity of the blood-stream must have been

ery mild. In all probability both factors come into play. The disturbance of the sympathetic influence upon the cord vessels might be held responsible for a dilatation and increased permeability of their walls, so facilitating the passage of toxins into the surrounding tissues. Evidence of this is seen in the proliferation of the perivascular neuroglia. It is conceivable, however, that with time a permanent paresis of the vessel wall with slowing of the blood-current occurs, thus affording the most favourable conditions under which mild toxicity of the blood could exercise a deleterious action upon the vessel walls and nutrition of the nervous tissues generally. Whether we are right or not in attributing importance to the sympathetic factor must be left to further experiment. Our present position with regard to these hæmatogenous lesions is that the term "general intoxication" expresses their pathology inadequately.

From the above clinical and experimental study it is clear that the two mechanisms of infection of the cerebro-spinal system—the hæmatogenous and lymphogenous—are characterised by sufficiently distinct morbid phenomena, and if we apply the results of the experiments to the human subject we obtain very considerable assistance in arriving at an understanding of the genesis of certain lesions.

We have brought forward ample evidence to show that acute and chronic myelitic conditions are readily produced by infection of the ascending lymph system in nerves. We have previously given it as our opinion that general paralysis of the insane is a chronic inflammatory disease of lymphogenous origin. This opinion is based on the close similarity between the vascular lesions in this condition and those found in our experiments where the lymph system of the nerves or cord is infected. The striking predominance of adventitial proliferation and infiltration can be explained only by toxi-infection of the cerebro-spinal lymph. There is no evidence of a general blood intoxication, for in dementia paralytica, as in our experiments, the endothelium of the vessels may be quite unaffected, while the adventitial spaces are packed with the products of proliferation. Further, to tabes dorsalis we assign the same lymphogenous genesis. The vascular phenomena, similar to those in general paralysis, the constant primary affection of the root entry zones, and the rigidly systemic character of the lesion preclude any other conclusion.

We have shown that certain cases of acute meningo-myelitis fall into the lymphogenous category, and there is now a preponder-

ance of evidence to show that acute poliomyelitis must also be included in this group. The whole picture of this disease is one of a disseminated meningo-myelitis, and as the brain may be affected in adults especially, the more comprehensive term of disseminated meningo-myelo-encephalitis has been suggested by Wickmann. There are many facts which show that infantile paralysis cannot be a blood infection, and indisputable evidence in favour of the lymphogenous genesis. This latter view is upheld on clinical and experimental grounds by both Wickmann and Römer, and with them we agree. The localisation and morphology of the lesions and the continuity of extension are characteristic of lymphogenous infections, a continuity which varies naturally, and attains its maximum in the acute ascending paralysis of Landry.

There is one important feature in connection with the question of lymphogenous propagation and extension to which Homén has drawn attention, viz. that the posterior root ganglia act as traps or filters, and form a kind of barrier against the further centripetal passage of organisms. This observation, although difficult to explain at present, would seem to be of special significance, as in all our experiments dealing with lymphogenous infection we have found a large collection of reaction cells and haemorrhage at the proximal pole of the ganglion. It may be that this special reaction indicates the existence of some defence mechanism against the further central extension of toxi-infection, and that an additional factor is introduced by a local anatomical structure. It is of interest to note in this connection that an analogous though more intense lesion is found in the ganglion in herpes zoster, and we would suggest that this disease also falls into the lymphogenous group, for we have observed the typical clinical appearances and pathological lesions of zoster to follow an empyema accompanied by ascending inflammation of the intercostal nerves. It is not improbable, therefore, that Homén is quite correct in calling the root ganglion a trap for noxious agents ascending in the lymph stream of nerves; and if we accept that it is a defence mechanism, and herpes zoster an expression of arrested ascending lymphogenous infection, then the suggestion might be put forward that the exciting agent which is not trapped passes onwards to the spinal cord and causes meningo-myelitis in some form or other.

A consideration of the phenomena in the subacute non-systemic lesions of the cord, such as occur with or without anaemia, Addison's disease, cancer cachexia, etc., shows that they must be included in the haematogenous category. There is an entire absence of the

proliferative change in the adventitia of the veins and capillaries which characterises the lymphogenous infections. The root entry zones in the posterior columns are, except perhaps in the latest stages of the affection, quite sound, while there is a marked sclerosis around the postero-median septum. The nerve-cells in the grey matter maintain their integrity. The morbid picture is not inflammatory in type, and corresponds with what is found in experimental hæmatogenous intoxication.

CLINICAL STUDIES. X.—FAMILIAL CIRRHOSIS OF THE LIVER: FOUR CASES OF ACUTE FATAL CIRRHOSIS OF THE LIVER IN THE SAME FAMILY, THE PATIENTS BEING RESPECTIVELY NINE, TEN, FOURTEEN, AND FOURTEEN YEARS OF AGE; SUGGESTED RELATIONSHIP TO WILSON'S PROGRESSIVE DEGENERATION OF THE LENTICULAR NUCLEUS.

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In the year 1912 my old house-physician and friend, Dr. Kinnier Wilson, published a remarkable monograph on a new disease which he termed "progressive lenticular degeneration: a familial nervous disease associated with cirrhosis of the liver."* "Progressive lenticular degeneration may," he says, "be defined as a disease which occurs in young people, which is often familial but not congenital or hereditary; it is essentially and chiefly a disease of the extra-pyramidal motor system, and is characterised by involuntary movements, usually of the nature of tremor, dysarthria, dysphagia, muscular weakness, spasticity, and contractures with progressive emaciation; with these may be associated emotionalism and certain symptoms of a mental nature. It is progressive, and, after a longer or shorter period, fatal. Pathologically it is characterised predominantly by bilateral degeneration of the lenticular nucleus, and in addition cirrhosis of the liver is constantly found, the latter morbid condition rarely, if ever, giving rise to symptoms during the life of the patient."

Dr. Wilson analysed 12 cases, 4 of which were observed by himself. Of these cases the age of the youngest at onset was 10 years, and of the oldest 26 years. The average age, calculated from the series of 12 cases, is 15. In these 12 cases, no fewer than 8 were familial cases.

In considering the nature and pathogenesis of the disease he concluded that—

1. It seems certain that the disease is not due to a congenital or abiotrophic defect.

* *Brain*, vol. xxxiv. part iv. p. 295.

2. The presumption therefore is strong that the disease is acquired.

3. There is evidence to show that the disease is toxic in origin, but none to suggest that this toxin is syphilitic.

4. It is possible this toxin may be elaborated in the liver.

5. The toxin has a specific action on the lenticular nucleus.

6. The nature of the toxin is unknown; it is almost certainly not microbial.

My present purpose is to record a remarkable series of cases of *acute* cirrhosis of the liver, due neither to syphilis nor to alcohol, occurring in one family, and to suggest their relationship to cases of progressive lenticular degeneration.

Dr. Wilson states that in his cases of progressive lenticular degeneration the cirrhosis of the liver which was constantly found rarely, if ever, gave rise to symptoms during the life of the patient. In this respect, and also in the fact that in the series of cases of familial hepatic cirrhosis which I am about to record, no nervous symptoms were observed, there is an essential difference from Wilson's cases of progressive lenticular degeneration. Nevertheless I am disposed to think that the two conditions are probably related. My suggestion is that familial cirrhosis of the liver without nervous symptoms is perhaps merely a preliminary and not fully developed stage of the disease which Wilson has termed progressive lenticular degeneration. If, as Wilson concludes, the nervous lesion (the lenticular degeneration) is probably due to a toxin generated in the liver, the result of the cirrhotic condition, it is not difficult to suppose that cases of familial cirrhosis of the liver, due to the same cause, whatever that may be, as the cirrhosis of the liver in progressive lenticular degeneration, may from time to time occur without the development of lenticular degeneration and nervous symptoms. In this connection it is most important to note that in my series of cases the course was very acute – the duration of the symptoms in each of the 4 cases was three or four weeks—whereas the duration of the 3 most rapidly fatal cases of progressive lenticular degeneration described by Wilson was four, six, and thirteen months respectively; while the shortest of the more chronic cases lasted two and a half years, the longest for seven years. The average duration of the first 3 of Wilson's own cases was three and a half years.

The details of the series of cases of familial cirrhosis of the liver to which I now wish to refer are as follows:—

Case I.—H. M., a boy, aged 9, was seen in consultation on 5th June 1899 suffering from ascites.

History.—The history was that for a week or two he had not been well, but had been at school until Thursday, 1st June 1899. On Friday, 2nd June, he found that his trousers would not meet owing to swelling of the abdomen. He had had no pain, no cough, no shivering, but the temperature was found to be 101° and the pulse 110.

When I saw him the abdomen was considerably distended with fluid. The abdominal parietes were œdematous, the scrotum was swollen, the feet and legs were not swollen, the face was slightly yellow, the conjunctivæ were distinctly jaundiced. The pulse was 110, the temperature 101°. The tongue was covered by a white fur in the centre and was red at the edges. Owing to the distended condition of the abdomen the size of the liver and spleen could not be ascertained. The heart and lungs were normal. The urine contained some bile pigment but no albumen or sugar.

Subsequent Progress of the Case.—*On 12th June* 1899 the abdomen measured 27¾ inches and contained a large quantity of fluid. The jaundice was rather deeper than it had been. The temperature was 99·6°, the pulse 108. There was no headache and no vomiting, and there had been none since the commencement of the attack.

Thinking that the case might be one of acute yellow atrophy of the liver a specimen of urine was sent on this day to the Royal College of Physicians' Laboratory, and Dr. Noël Paton reported that "after it had been evaporated and extracted with alcohol, it gave a large crop of tyrosin crystals but no leucin crystals."

The blood was also carefully examined the same day by Dr. Lovell Gulland. His report was as follows:—"Blood dark, 'sticky,' does not coagulate quickly. A fresh specimen examined microscopically showed rouleaux formation imperfect. Corpuscles have a very marked tendency to become crenated both in wet and dry films. Blood plates few, fibrin slight and delayed. Red blood corpuscles 5,184,000, and white blood corpuscles 18,600, per cubic millimetre, hæmoglobin 57 per cent. Stained films show marked crenation of the red corpuscles, some of which are small and contain less hæmoglobin than normal. No nucleated reds seen. The leucocytes were mainly polymorphs. A few blood-plates were present. No micro-organisms were found in the blood after careful staining."

On 19th June the abdomen, which measured 29 inches, was tapped and a Winchester quart of fluid drawn off. The spleen could not be felt. After the abdomen had been emptied the liver dulness was found to measure 2 inches. The patient had become thinner. A few petechial hæmorrhages were present over the abdomen.

On 20th June the patient suddenly collapsed; the pupils became widely dilated: he got very restless, apparently as the result of

cerebral anæmia: the pulse was thready and uncountable. He died on the afternoon of this day.

Post Mortem Examination.—This was made on 21st June at 10 A.M. by myself, in the presence of Drs. Lovell Gulland and Veitch.

The abdomen contained a considerable quantity of fluid; a few flakes of recent lymph were found over the intestine. There were numerous small petechial hæmorrhages on the surface of the large intestine and on the outer surface of the heart. The stomach contained some coffee-grounds fluid.

Some of the lymphatic glands in the abdomen were enlarged.

The liver was a typical example of "hob-nailed" cirrhosis.

The spleen was enlarged and congested. The lungs and kidneys were congested. The suprarenals were normal. The bone marrow was slimy and very red.

Microscopic examination of the liver, which was made by Dr. Gulland, showed that it was a typical example of "hob-nailed" cirrhosis.

Remarks.—Cirrhosis of the liver occasionally occurs in young subjects, but it is rare. In some of these juvenile cases the cirrhosis is no doubt due to alcohol; in others to syphilis; but in some cases there is no apparent cause.

Careful inquiry in this particular case enabled us to exclude any suspicion of alcohol and syphilis; the parents were strictly sober people, the children were always well fed, well clothed, and well looked after, and none of them had ever been in the habit of taking alcohol. In this respect, therefore, the case is of considerable interest, but the chief point of interest in the case, and it is a very remarkable one, is that *three other members of the family* (three sisters) *had apparently died from the same condition.*

Family History.—The details are as follows:—The father, aged 47, and the mother, aged 42, are strong, healthy people. On the father's side one brother died of phthisis, on the mother's side one brother committed suicide, another was melancholic.

There was no evidence to show that the father, mother, or any of the children had suffered from syphilis—there had been no miscarriages, and there were no indications of inherited syphilis, so far as I could learn, in any of the children.

The family consisted of seven children.

1. A male, aged 23, is alive and well, thin, but wiry.

2. A girl, aged 10, *died apparently from the same disease—cirrhosis of the liver*; a year before her death she was knocked over at school and was laid up for some time after this with a swollen knee: her fatal illness lasted *two or three weeks.*

3. *A girl, aged* 14, *died apparently from the same disease—cirrhosis of the liver. She had previously suffered from joint rheumatism; her last illness lasted three or four weeks.*

4. *A girl, aged* 14, *died from the same disease. I personally saw this patient in consultation; she was sent to me on* 17*th May* 1898. *The abdomen was much distended with fluid, and the patient was jaundiced. There was no pain and no evidence of tuberculous disease in the abdomen or liver. The urine contained bile pigment and a large quantity of uric acid. The disease advanced with great rapidity, and the patient died on* 23*rd May* 1898.

5. A boy, aged 11, was puny in infancy, but is now healthy.

6. *The patient.*

7. A boy, aged 7, quite healthy.

Dr. Proudfoot of Kirkcaldy, the ordinary medical attendant of the family, wrote me that "*all the cases presented the same general symptoms—rise in temperature, jaundice and ascites, death in the course of three or four weeks.*"

Isolated cases of cirrhosis of the liver are occasionally, though rarely, met with in young subjects in whom there is no suspicion of alcohol or syphilis, and for which there is no apparent cause. A few cases of this kind have, in my long experience, come under my notice. I suggest that some of these isolated cases are, perhaps, of the same nature as the familial cases described above; and perhaps in reality the same disease as Wilson's progressive lenticular degeneration. Everyone, of course, knows that in every familial disease, such, for example, as pseudohypertrophic paralysis, isolated cases are of frequent occurrence. Further, the true nature of such isolated cases is, in rare familial affections, apt to escape notice; while the mere occurrence of several cases of the same sort in one family always attracts attention and makes the diagnosis much more easy.

The following is a typical and striking case of cirrhosis of the liver, in a young subject, in which there was no history of alcohol, no suspicion of syphilis, and for which there was no apparent cause. I suggest that it was perhaps of the same nature as the familial cases related above, and possibly a preliminary stage of Wilson's progressive lenticular degeneration:—

Case II. —*Cirrhosis of the Liver in an Unmarried Woman, aged* 20; *no Suspicion of Alcohol or Syphilis; no Apparent Cause.*—M. B., female, aged 20, was admitted to the Edinburgh Royal Infirmary on 21st March, and died on 15th June 1894.

Previous History. — *Three days before the patient's admission to the*

February she strained herself while lifting a large tub 2½ feet in diameter, which was half full of water, from the floor to a stand. As the result of the strain she immediately felt a severe pain in the right hypochondrium, and a feeling of nausea and faintness. After a short rest she went on with the washing on which she was engaged. The next day she felt better, but the pain still troubled her when she made any movement.

She told me quite emphatically that until the strain she was quite well and had had a good appetite.

On 12*th March* she noticed that her feet were swollen in the morning, and that her eyes were puffy. On this day she felt sick, but did not vomit. During the course of the day she was breathless; her appetite and digestion became impaired: her condition was so serious that she was immediately sent to the Infirmary by the doctor whom she consulted.

The patient had had measles and scarlet fever in childhood, but never rheumatism nor chorea.

Family History.—The patient is the eighth child in a family of nine. Five of her brothers and sisters are dead. Her elder brother, who is aged 36, is delicate; a sister younger than the patient died at the age of 13 of heart disease and dropsy; the causes of death of the other brothers and sisters could not be ascertained. The mother suffers from heart disease, and the father is rheumatic.

Condition on Admission.—On examination the patient was found to be somewhat anæmic. The face had a puffy, flabby, swollen appearance; the feet and legs were œdematous; the abdomen was distended but tympanitic. The appearance of the patient was highly suggestive of kidney disease; but the urine, which was repeatedly examined, never (until the final stages of the case, and then only for a few days) contained either albumen or casts. The tongue was large and flabby, and coated with a yellow fur. The breath was fœtid. The patient complained of a bad taste in the mouth, and of pain after eating. She lay with her legs drawn up, and there was localised tenderness in the right side of the epigastric and the right hypochondriac regions. This pain was very localised. The bowels were constipated. The abdomen was very tensely distended, apparently as the result of spasm of the abdominal muscles. The superficial veins were not prominent. The umbilicus was flush with the anterior abdominal wall. Percussion yielded a tympanitic note all over the abdomen, which did not at this time, so far as we could detect, contain any fluid.

The liver dulness was normal. The upper part of the abdomen was more distended than the lower, and the distention was clearly due to a large dilatation of the stomach. The shape of the organ could be very readily determined both by inspection and percussion.

The localised tender spot measured 1 inch square; it seemed to be situated just about the position of the portal fissure and pyloric end of the stomach.

Micturition was frequent and painful. The amount of urine passed was normal; it was extremely dark in colour; specific gravity, 1022; it contained phosphates and a large quantity of indican, no albumen, no bile, no casts.

The patient stated that she was very short of breath on exertion. This appeared to be the result, partly of anæmia, and partly of the tense distension of the abdomen and the upward pressure of the diaphram.

The heart appeared to be normal. The pulse was full and regular —80 per minute.

The patient had not menstruated for three months; before this she was quite regular.

Two days after the patient's admission to hospital she vomited; the vomited matters consisted of undigested food and were bile-stained.

Diagnosis.—The case was a very obscure one, and we watched it carefully for some time and repeatedly examined it. It was obvious that the stomach was greatly dilated, and it seemed probable that there was a localised peritonitis in the region of the pylorus. I was disposed to think that the facts of the case were best explained by supposing that a localised peritonitis had been produced by some rupture at the time of the strain, and that the pressure of the adhesions was the cause of the dilatation of the stomach. The possibility of the case being one in which an ulcer near the pyloric orifice had previously existed without marked symptoms was kept in view. If such an ulcer had been present, with adhesions tying the outer surface of the stomach to the liver or adjacent parts, the strain might, it was thought, have ruptured some of these adhesions. In this way the symptoms might perhaps have been explained.

Treatment and Progress of the Case.—The patient was carefully dieted. On several occasions an attempt was made to wash out the stomach, but this plan of treatment had to be discontinued. The patient rebelled most forcibly against the introduction of the instrument. Whenever the tube was introduced into the stomach it was immediately shot out with extreme expulsive force. The violent muscular contractions produced in the stomach were very remarkable and were attended with great distress, and on more than one occasion by the discharge of a small quantity of blood.

A mixture containing bicarbonate of potash, bicarbonate of soda, aromatic spirits of ammonia, tincture of rhubarb, and infusion of calumba was prescribed.

On 28*th March* the œdema was very much diminished, the tongue was rather cleaner, and the patient's condition seemed to be slightly improved.

On 16*th April* it was noted — "The abdomen is still greatly distended and contains a good deal of fluid. The stomach is greatly dilated, tensely distended and tympanitic, as it has been since the patient's admission. She complains of pain over the region of the liver and in the right hypochondrium generally. The tongue is slightly furred. Dr. Barbour, who kindly examined the patient to-day (the possibility of pregnancy had been kept in view), reports that the pelvic organs are normal.

On 17*th April* the abdomen was aspirated midway between the umbilicus and the pubes, but no fluid was withdrawn. During the succeeding night the patient had a slight hæmatemesis.

On 18*th April* she complained of more pain over the region of the liver.

On 2*nd May* the patient vomited in the morning and complained of pain over the abdomen. The ascites was much more marked.

On 9*th May* the patient was tapped and 232 ozs. of clear straw-coloured fluid withdrawn. After the tapping the patient complained of localised pain and tenderness at the old spot, about the pyloric region of the stomach.

On 18*th May* she was again tapped, and 229 ozs. of clear fluid withdrawn.

On 21*st May* the urine for the first time was found to contain a considerable quantity of albumen and some blood (the patient was not menstruating). The colour was still very dark. No tube casts were detected.

On 22*nd May* there was a slight epistaxis.

On 28*th May* the patient vomited a small quantity of bilious matter.

On 2*nd June* the abdomen was again tapped, and 255 ozs. of fluid withdrawn.

Notwithstanding the repeated tappings, the patient did not improve. She was consequently transferred to the surgical side of the hospital, with the object of having an exploratory incision made into the abdomen; it was thought that there might possibly be some lesion in the region of the portal fissure which would permit of successful surgical interference.

The operation was performed on 14*th June*; the liver was found to be small and shrunken; there was nothing which could be removed by the surgeon. During the course of the night the patient vomited a

considerable quantity of coffee-grounds material. The vomiting was followed by collapse from which she never rallied. She died on the evening of the 15*th*.

Post-Mortem Appearances.—The post-mortem examination was made on 18th June. The body was distinctly jaundiced; this jaundice had developed during the last day of her life. The stomach was considerably dilated.

The liver was a typical example of the ordinary "hob-nailed" form of cirrhosis. It weighed 3 lbs. 7 ozs. Firm, old adhesions of some length extended between the liver and the gall-bladder and the adjacent parts (costal margin, pyloric end of the stomach, and transverse colon).

The gall-bladder contained a quantity of thick bile which was becoming transformed into a pasty mass.

The spleen was very large; it weighed 1 lb. 8½ ozs. and measured 8 ins. by 5 ins. in breadth. It was of firm consistency and dark-coloured, but it presented no other change than that due to chronic venous congestion.

Three or four submucous haemorrhages, the largest about the size of a sixpence, were present in the posterior wall of the stomach. There was no ulceration. The mucous membrane was markedly thickened, granular and pigmented, indicative of chronic venous engorgement and chronic catarrh. There was no appearance of an old ulcer; nothing at the pylorus, except the adhesions which tied the stomach to the gall-bladder and liver, to account for the dilatation.

The kidneys weighed 4 ozs. respectively; they were bile-stained, and showed appearances of commencing decomposition, but were otherwise normal.

Remarks.—The case presents several points of interest, both from a pathological and clinical point of view. The liver was a typical example of the ordinary common form of "hob-nailed" cirrhosis—a condition which is seldom met with before the age of 25, and very rarely in children. A few years ago I met with a typical case in a girl, aged 11. The disease in that case seemed to have occurred after an attack of scarlet fever. In some juvenile cases alcohol is, no doubt, the cause. In the case recorded above the patient's father, mother, and brother were alcoholic; but the most careful inquiry failed to show that the patient herself was addicted to the use of alcohol; in fact, the contrary seems to have been the case; she is said to have had a dislike and objection to liquor in any form.

The diagnosis was most difficult. I have already referred to this point.

Another clinical point of interest was that at the time of the patient's admission to hospital the feet and face were œdematous. The patient was very pale and puffy, and the appearance exactly resembled that due to kidney disease. At first sight I could hardly bring myself to believe that the case was not one of Bright's disease. I repeatedly examined the urine myself, and on no single occasion found albumen. The urine, however, contained a very large excess of indican. This was a clinical feature of some interest.

The possible relationship of cases of familial cirrhosis of the liver and Wilson's progressive lenticular degeneration has already been referred to.

THE BIRTH-RATE COMMISSION'S REPORT AND EVIDENCE.

In order to estimate correctly the importance and value of the evidence and the *Report* * furnished by the National Birth-Rate Commission certain facts must be kept clearly in mind. In the *first* place the Commission commenced work in the close of 1913, and was therefore going on before the war began. This circumstance is noteworthy, for the tremendous destruction of adult life caused by the gigantic armed struggle going on in Europe, accompanied as it has been in some places by an increase in infantile mortality, has perforce altered current views on the significance of a falling birth-rate. It is hardly going beyond known facts to assert that many people who took little notice of the decrease in the birth-rate in 1913 are paying attention now in 1916, and that others whose interest then was slight are now keenly alive to the paramount importance of the phenomenon. Nothing less than a marked shifting of public interest and emphasis has occurred during the past two years. In the *second* place the Commission of Inquiry was established by the *National Council of Public Morals for Great and Greater Britain*, a body which has for its object the spiritual, moral, and physical regeneration of the race. This fact must not be lost sight of, for it serves to explain to some extent the methods of inquiry adopted by the Commission, the uses to which it put the information which it obtained, and the form in which it drew up its conclusions. The National Council has a Branch Scottish Council for Public Morals, under whose auspices a conference was held in Edinburgh on 21st October 1913, when addresses were given by Mr. James Marchant, F.R.S., Edinburgh (the Secretary of the National Council and Editor of the present Report), by Dr. J. W. Ballantyne, by Dr. C. W. Saleeby, and by Professor J. Arthur Thomson, and when the composition of the Commission and the plan and scope of its inquiries were announced and explained. In the *third* place the members of the Commission were chosen so as to represent the various classes and groups of people who might be supposed to be interested in the subject of the falling birth-rate, and who might

* *The Decline in Birth-Rate. Its Causes and Effects.* Being the Report of, and the chief evidence taken by, the National Birth-Rate Commission, pp. 450. London: Chapman & Hall, Ltd., 1916.

book, opinions regarding its significance and the methods of dealing with it—the clergy, the medical profession (including women doctors), statisticians, publicists, and some members of the aristocracy, as well as Mrs. General Booth of the Salvation Army, had seats on the Board and brought their own particular contributions to the discussions. Further, the Commission gained a certain amount of semi-official importance by the presence on it of Dr. Newsholme of the Local Government Board and of Dr. T. H. C. Stevenson, the Superintendent of Statistics for the General Register Office, as well as by the announcement which was made in the House of Commons by Mr. Asquith, that no Royal Commission would be appointed, and that the Government would await the present report with interest. The other medical members were Sir J. Crichton-Browne, Sir Malcolm Morris, Sir H. B. Donkin, Professor G. S. Woodhead, Dr. Major Greenwood, Dr. T. B. Hyslop, Dr. J. W. Ballantyne, Lieutenant-Colonel F. Fremantle, Dr. A. T. Schofield, Dr. C. W. Saleeby, Dr. Mary Scharlieb, Dr. Florence Willey (Lady Barrett), Dr. Agnes Savill, and Dr. Ettie Sayer. In the *fourth* place the Report is intended for the reading not of any one group of people but for the thinking and understanding public of all classes and professions. Consequently neither the medical profession, nor the clergy, nor any other single class of society, must expect the matters dealt with to be surveyed from their sole standpoint, and they must allow for this in any comments or criticism they may feel inclined to make. The time, therefore, when the Commission met, its origin, its status, its composition, and the public to whom its Report is addressed must all be kept prominently in view in gauging the value and the results of its work. The Commission was fortunate in having Bishop Boyd Carpenter, K.C.V.O., as its Chairman for two years, and thereafter the Very Rev. Dean Inge, D.D., presided at the meetings. At first the Commission sat at 6 Little Cloisters, Westminster Abbey, and later at the Deanery, St. Paul's.

Certain general conclusions can at once be reached regarding the results of the Commission's work. In the *first* place the inquiry was evidently a full one, and it is only necessary to read the evidence in order to realise that no trouble was spared in eliciting the real views of qualified experts. In the *second* place, and as a consequence of this, the evidence contains, even now after it has been edited, many matters which are not usually published in non-medical works: but it is to be observed, and the members of the Commission deserve thanks for it, that the Report itself is

very carefully worded, and that its conclusions are stated with fulness and yet with an amount of restraint which has withdrawn from them anything which the prurient mind could lay hold upon for evil. This is no small accomplishment, and it is in itself a justification of the existence and the work of the Commission. In the *third* place the Report supplies an authoritative statement of the essential facts of a vastly important subject of national interest: the data regarding the falling birth-rate in this country are no longer in doubt, and the Report can be consulted with assurance upon the many questions involved. In the *fourth* place the Report to a certain extent attempts to apply the facts and to draw some conclusions of a practical kind from them; but in the nature of the case it was somewhat hampered in this direction, for, of course, constructive schemes must depend on the view-point of the individual members of the Body of Inquiry, and this was not always the same. Consequently it is clear that much of the help to be derived from the Report must be got by each body of interested persons drawing from the facts contained in it the conclusions specially pertaining to their kind of activity and sphere of thought. The Report will thus serve as a quarry from which stones can be had for the building of edifices of various kinds. For instance, the Church will look at it from its standpoint, the medical profession will survey it from its side, the social economist from his, the moralist from his, the historian from his, and the politician from his. The Report, therefore, marks rather a beginning than an end: it gives with assurance certain data; it attempts somewhat tentatively to apply them to large outstanding problems of Church, State, medicine, morals, etc., and is forced into giving a *Report*, an *Addition* to the Report, and a *Reservation*; and it provides for the future materials from which constructive schemes of social reform, of medical advance in the prevention of disease, and of reorganisation and readjustment of clerical outlook upon and possibly of interference with the individual conscience in relation to matters of reproduction.

The first section of the Report deals with *statistical evidence*, and the Commission found itself warranted in concluding that in Great Britain, as in the other countries of Northern and Western Europe, there was unmistakable proof that a sharp decline was taking place in the birth-rate. Further, there seemed no reasonable ground for doubting that the countries included would ultimately reach the State of France. In Italy, in Eastern and in South-Eastern Europe, there was, on the other hand, little or

no evidence of a declining birth-rate. The birth-rate in England and Wales had declined approximately to the extent of one-third in the last thirty-five years—from 36·3 in 1876 to 23·8 in 1912. There had been, it was true, a slight rise in the rate prior to 1876, but this was to be regarded as due to the more rigid enforcement of registration rather than to any real increase in fertility at that time. The birth-rate from 1840 to 1880 might be put at the steady figure of 35 per 1000 population. The figures given seemed also to show that in the countries, such as France, in which the decline had first showed itself the downward progress had been slow, whilst in those in which it had been recent, such as Germany, the fall had been more precipitate. It was understood, of course, that migration and other factors (*e.g.* efficiency of registration, etc.) causing variation were taken into account in forming these opinions.

There had been, therefore, a Northern and Western European decline, and that had swept in Great Britain—but not Ireland, or at any rate not the Ireland which lay outside of Ulster. That is a striking and an arresting fact. The Report says: "Adopting a standardised or corrected rate it was found that the rate in Connaught in 1911 was 45·3 as against 24·7 for England and Wales, and the fertility in Connaught had been rising in the last few years with even greater rapidity than it had been declining in this country; the increase in Connaught had been shared by the other provinces with the exception of Ulster, where there had been a decrease of slight extent; for Ireland as a whole the rate rose from 36·1 to 37, while that for England and Wales fell from 28·4 to 24·7 and for Scotland from 33·4 to 29·7." The Commission, which was always very shy of expressing opinions, quoted in relation to the Irish figures the view of Dr. Stevenson, that artificial restraint had become more prevalent in England, as in other similarly situated countries, whereas the Irish increase represented the natural result of increased prosperity in the case of a population amongst whom the religious bar to such restraint was effective. Whether this be so or not it will be well worth the while of Protestant, and especially of Presbyterian communities, to study closely the relation of the Roman Catholic teaching to the value placed upon antenatal life and to the duties of married people in matters of reproduction.

In this statistical part of the Report it would seem as if the Commission tried hard to find other causes than artificial restraint to account sufficiently for the decline in the birth-rate. They

were led, however, to the conclusion that the fall was not due, to any important extent, to alterations in the marriage rate, to a rise of the mean age at marriage, or to other causes diminishing the proportion of married women of fertile age in the population. Density of population was insufficient to explain it, for the rural rate was not widely different from the urban; food might have an influence, but it would be difficult to get facts or figures of any value; the theory of cyclical variations in the natural power to conceive or to procreate, the hypothesis of variability of germinal vitality, as stated by Dr. Chalmers of Glasgow and others, was very attractive, but it broke down in face of the Irish figures. It seems almost as if the Commission reluctantly gave up the view that the higher education of women had something to do with the decline; at any rate it has to be abandoned, for statistics seem to show that there is no physiological difference between the fertility of college and non-college women, although there surely must be a slight difference in the probability of babies being born due to the later age at which college-bred women must, almost of necessity, marry. Although no sufficient cause for the falling rate could be discovered outside of conscious limitation and artificial restraint, yet the following circumstances did affect fertility, and brought it about that the decline must be regarded as dysgenic and not eugenic, as will be evident. The fall in the birth-rate in England and Wales had been more marked in those districts in which a higher standard of living was found; the size of family tended to vary inversely as the social status of the parents—professional and allied occupations having low fertility, and labouring occupations generally a high one (that of coal miners being very nearly twice that of doctors); housing conditions had an influence, for fertility decreased regularly as the size of the tenement increased—one- to three-roomed houses giving more births than six- to seven-roomed ones—and infantile mortality decreased along with it; but the saving of infant life in more comfortable tenements compensated only to a slight extent for their lower fertility. Thus, to sum up, it must be admitted that fertility is closely connected with social status, the relation being such that the more prosperous the social class the lower is the fertility; more than that, there is no proof that the higher mortality among the infants of the lower social class suffices to adjust the difference. The decline, therefore, is not eugenic, cannot be conceived of as in any sense a good thing, but is out and out dysgenic. The babies are, so to say, not fewer and of

finer quality; they are fewer and are more from the lower social strata.

Allusion is made to the voluntary census of the Fabian Society, and although conclusions from it can hardly be regarded as of great value in respect of fertility, yet it does suggest that conscious limitation is widely practised among the middle and upper classes. The Commission also made investigations of its own on this subject, and obtained the rather unexpected result that the average *size* of family was rather greater among those who adopted conscious methods of restriction than among the others, but childless marriages were excluded from the figures in both cases. The furthest length that the *Report*, as distinguished from the *Addition*, is able to go is to state "upon evidence less substantial" than that on which the conclusions respecting the distribution of the decline depend, that "conscious limitation of fertility is widely practised among the middle and upper classes, and there is good reason to think that, in addition to other means of limitation, the illegal induction of abortion frequently occurs among the industrial population." The Addition to the Report, as will be seen, goes further, and expresses an opinion on the regrettability of the decline.

The second section of the Report deals with certain *economic and social aspects* of the subject very clearly and very pithily. It deserves careful study. It discusses the questions of rapidly increasing, stationary, and decreasing populations; and it concludes that, for a country open to commercial intercourse with the world, like Great Britain, the law of Malthus (that population tends to press upon the means of subsistence) does not necessarily hold at all, although for a closed country, which must produce all its own material necessaries, it holds good as a general proposition. There is no reason to think that a further reduction in the English birth-rate would at the present time give a larger yield of wealth per head. Labour and Capital and the World Standpoint are very difficult subjects, but "we must face the fact that every rise in the condition of the artisan tends at present to lower the birth-rate in his class," a somewhat significant matter in relation to wages. Of general over-population, from the Work Standpoint, in any absolute sense of the term there was no evidence. As to the quality of the population it was impossible to make definite statements; but there was recent evidence to indicate that the supposed differences in inherent racial qualities were much slighter than had been alleged, and that education and

cultural environment explained the greater part of what had been considered to be ethnical differences. A pressure of population in any country, however, was a chief cause of international disputes and wars; and war in its effect upon the quality of a population was generally admitted to have a selective power which was dysgenic. The Commission trod lightly over the vexed question of class inferiority, but thought that most of it was due to bad environment, and deprecated "the tendency to identify the economic *élite* with the pyscho-physical *élite*." This is one of several instances in the Report in which there was a traceable inclination to give an inferior position to heredity and eugenics as contrasted with the influence of environment and education. The Commission could not accept "the hypothesis that the broad distinctions between social classes were but the effects of germinal variations."

The *Housing question* occupied the third section of the Report, and was another matter about which the Commission found it extremely difficult to make any recommendation. Children were the crux of the question; and landlords did not willingly cater for large families; in fact, they preferred childless tenants. The reasons were not hard to find; and so it came about that sometimes the mere probability of the arrival of another child brought the anxiety of a possible eviction. The housing question, both in town and country, made the rearing of large families by the working classes a matter of great difficulty, and also affected the birth-rate. Cheap and rapid transit from large cities to outlying districts in some degree lessened this difficulty.

The *medical aspects* of the declining birth-rate are stated in the fourth section of the Report with admirable restraint (since the general public were to be invited to read), and yet with complete understanding. The Commission wisely pointed out that generalisations should be condemned which took no cognisance of the medical, medico-legal, and ethical distinctions between the different forms of conscious limitation (restriction of coitus to certain times, use of diachylon pills, sterilising operations, and criminal induction of abortion), each of which reduced the birth-rate. It was unable to give a definite pronouncement as to the physical consequences of the use of contraceptive methods: the evidence was insufficient. The Commission joined with the Law and with Medical Ethics in condemning the destruction of the product of conception, save where the life of the mother was at stake; and Monsignor Brown put a *Note of Reservation* to the

Report, in which he stated that his Church (the Roman Catholic) forbade it, even when the life of the mother was at stake. The Commission fully realised that in all such cases of destruction *a fortiori* was lost—that of the embryo or unborn infant; there was also risk to the mother's life. It thought that diachylon plaster should either cease altogether to be manufactured, or that it should be scheduled as a poison. The paragraphs on the effects of gonorrhœa and syphilis respectively on the birth-rate were models of clear exposition and accuracy, yet made generally and unobjectionably intelligent to the non-medical reader. The Commission, however, left the medical section of its report rather without conclusions; but these were to some extent supplied by the *Addition* which was signed by the large majority of the members. Of course the purpose of the Commission was to inquire if the birth-rate had fallen, and why, rather than to construct means to raise it again.

The *religious and moral aspects* were summarised in the fifth section. With regard to the former, the views of the Church of England, the Roman Catholic Church, some of the Free Churches, and the Jews were given, and they were all at one in their condemnation of and opposition to, not only the practice of abortion, but also to the prevention of conception by mechanical and chemical means, although the opinions and practice of the Free Churches seemed to lack definition. The Presbyterian or Church of Scotland standpoint was not separately dealt with. One has the feeling from reading the evidence which follows the Report that in the Free Churches—and one may probably add the Presbyterian Church of Scotland—a great deal of reticence has been shown by the clergy in expressing the opinions they hold. It would undoubtedly be well if all the churches made themselves acquainted with the Roman Catholic procedure and standpoint as stated by Monsignor Brown (pp. 389-413), with the Anglican as set forth in the 1913 Report of a Committee of Bishops (pp. 382-388), and with the Jewish (pp. 425-436) as explained by the Very Rev. the Chief Rabbi (Dr. J. H. Hertz).

The Commission rather avoids the direct expression of opinion on the moral aspects, and examines in turn certain commonly quoted ethical principles such as "be fruitful, and multiply," "do not attempt to outwit Nature," "all deliberate waste of the physical sources of human life (including therefore the use of preventives of conception) is wrong," "self-denial in the marriage state is good for mind and body," and "the public interest demands a

high birth-rate, private selfishness desires a small family": but the closing paragraph of this section of the Report and of the Report itself (as distinguished from the Addition) is worth quoting in full:—

> "While we have confined ourselves to discussing the moral and religious aspects of the declining birth-rate in respect of the married, we cannot close our eyes to the fact that the more widely spread knowledge of the means of preventing conception by the unmarried not only involves the removal of the prudential restraint on licence in sexual relations, but may affect the birth-rate in future in two ways—(1) Marriage with its responsibilities may be avoided, since sexual gratification is being obtained without any social obligations being incurred; (2) a practice begun before marriage may be continued after marriage."

The *Addition to the Report*, which has only three fewer signatures appended, when compared with the Report itself, goes a good deal further in respect of the statement of views, not only regarding the meaning of the falling birth-rate, but even touching the steps to be taken to arrest it or to make the best of it. It is inevitably more interesting than the almost too careful and too restrained Report. For instance, to the question, Is the present decline of our national birth-rate regrettable? the answer is clearly in the affirmative, with the perfectly admissible addition that it would be well not only to increase but to improve the population; and three sound reasons are furnished for giving the affirmative reply, viz.—(1) That human life in itself, under healthy conditions, is desirable and valuable; (2) that looking to the British Empire beyond our own shores the possibility of an increase along with improvement of the population is manifest; and (3) that it is well to strengthen all the circumstances which make the home, its life, its training, its joys, its religious sanction and approval, etc., more secure in the heart of the nation. The signers of the addition admit that the third reason is rather sentimental; but there will be many who will join with them in the particular piece of sentiment which longs for children in the home. The second part of the Addition is occupied with a series of suggestions as to changes in economic and social conditions, or in public opinion and general custom, which would aid not only the increase but also the improvement of the population. These include (1) the "minimum" or "living" wage

with insurance against unemployment; (2) State bonuses for families under certain conditions; (3) the separate computation of the means of husband and wife in the Income Tax, and remissions according to the number of children; (4) the increase in the facilities given for good cheap education; (5) better housing, with suitable sleeping accommodation and without exorbitant rents; (6) censure, on the one hand, of the recklessness of the poor in taking unlimited parental responsibility, and of the selfish luxury of the rich who shirk it altogether or in large measure; and (7) a stimulation of the popular sentiment regarding the value and sacredness of family life, along with a fairer distribution of the burden of parenthood between the mother and the father. Three other suggestions are made, for which at any rate it is likely that the medical part of the Commission (which was relatively strong) was responsible. The first is that women should be assured that the pains of childbearing can be mitigated. It may seem strange to us in Scotland, where chloroform has been to be had ever since 1847, that it should be necessary to state this; but it would seem that it has required the strange means known as "twilight sleep" to bring this matter of anæsthetics in midwifery before the expectant mothers of the South. The second suggestion does not contain the *word* Eugenics, but it calls for an increased knowledge respecting the ways in which quality as well as quantity may appear in the expected babies. The third suggestion is perhaps the most important of them all, viz., that infantile and child mortality should be checked by means taken not only after but before birth; in other words, by a maternity and child welfare scheme. Finally, the Addition to the Report urges that religious and moral teachers should consider more thoroughly the problems involved so as to be better able to give advice when asked, and that they should study in particular the following five questions:—(1) Is parenthood the only valid reason for marital relations? (2) What motives justify the restriction of the family? (3) Is any mode of restriction except voluntary abstinence from marital relations moral or religious? (4) How would voluntary abstinence affect the health, comfort, and happiness of the relations of husband and wife? and (5) How is the character of the children in a home likely to be affected by the numbers?

Nothing, or almost nothing, has been said about the 363 pages of evidence which form the rest of the Commission's published work. That the pruning knife could surely have been used more

widely and deeply will probably be admitted by everyone: and yet the fulness is not without a certain kind of value, for it restores to some extent the atmosphere of the discussions, many of which were extraordinarily interesting. Further, for the small band of deep thinkers on this immensely important subject, no word is superfluous; they at least will peruse the evidence from end to end. J. W. B.

CLINICAL RECORDS.

A CASE OF HYPERTROPHY OF THE LABIA MINORA.

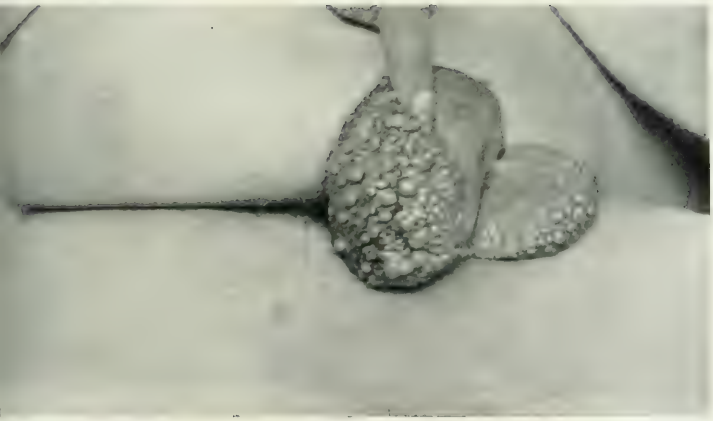
By DAVID M. GREIG, C.M., F.R.C.S.(Edin.).

Chronic hypertrophy of the labia minora leading to a permanent enlargement is not a common condition, but when it occurs it tends to assume appearances which are strikingly uniform. The patient, of which the accompanying photograph was taken, was an unmarried nullipara, aged 43. She had been the subject of cerebro-spinal sclerosis during the twenty-five years which preceded her death. The nervous disease was accompanied by increasing rigidity of the muscles, and latterly fixation, particularly of the lower limbs. From about the eighth year of her disease she had occasional retention of urine, and, as this became frequent, it resulted in overflow if the catheter were not daily employed. For many years she was the inmate of a hospital, and it is greatly to the credit of that nursing staff that no septic trouble ever resulted from the catheterisation. During the last few years of her life, when she was quite bedridden, the lower limbs were flexed and strongly adducted, and apparently to this was due the alterations in the labia. At no time was there ever any vaginal discharge, irritation of the vulva, or septic condition of the urine, but gradually the chronic congestion of the labia resulted in hypertrophy. The hypertrophy was of very slow growth, and had existed for years before the increasing size of the labia interfered with the manipulation of the catheter, and only then was my attention called to it. The pudendum appeared to be compressed by the extreme adduction of the flexed thighs. The labia majora and the perineum were normal. From between the former the labia minora depended in two prominent masses. The left was somewhat larger than the right. There was no undue moistness about the parts, and absolutely no sign of any previous inflammation. The exposed surfaces had assumed a nodulation, which is probably characteristic of this type of labial overgrowth, and—as is invariable when mucous membrane ceases to be protected and in contact with mucosa—had become dry and slightly darkened in colour, and the epithelium was obviously thickened. The tumours were

firm and could not be altered in size by pressure. They were neither tender to the touch nor painful. For the convenience of catheterisation the labia were removed—a very simple operation—with very satisfactory results.

The condition is that of chronic œdema of the labia, the result of interference with the lymphatic circulation of the parts. That it is lymphatic and not venous is proved by the fact that there is no alteration in the colour of the labia at their attachments, there is no telangiectasis, and nothing approaching varicosity. The condition has been named elephantiasis, a most objectionable term to my mind, as being associated with elephantiasis Arabum, a tropical disease with a pathology of its own. It is regrettable that, the striking feature of elephantiasis Arabum being increase in size, all conditions where enlargement is a feature have been indiscriminately called elephantiasis. Undoubtedly there is elephantiasis of the labia occurring where elephantiasis Arabum is endemic, but the condition now under consideration has no relation to elephantiasis. When the nymphæ normally project clear of the labia majora the exposed portions are dry and are pigmented, and the mucosa assumes the characters of epidermis. In the above case the appearances were merely a gross exaggeration of the normal, but along with them there was progressive increase in size. The nodulation is peculiar, and, I think, characteristic. The nodules were solid, though, at the first glance, some appeared cystic. An exactly similar condition, at any rate as regards one labium minus, is figured by Kelly.[1] That author calls it elephantiasis, and, in his first sentence of the description, says it "must not be confounded with elephantiasis." Surely he could have chosen another name and lessened the chance of the confusion he deprecates. He says negresses are specially susceptible to the condition, that the disease is of rapid growth, and that syphilis, leucorrhœa, or ulceration about the vulva are etiological factors. My case is sufficient to prove that the growth may be very slow, and in it neither syphilis, leucorrhœa, nor ulceration was present.

Pozzi,[2] who calls it "œdema of the vulva," also emphasises syphilis as a frequent precursor. Though not exactly the same as mine, but obviously a hypertrophy of the labia due to lymphatic obstruction, there is recorded by Croom[3] a case which proves the futility of the prevailing nomenclature. It is called "Elephantiasis Arabum Vulvæ," but there is not produced one item of evidence that it is elephantiasis Arabum; while the



Hypertrophy of Labial Mucosa showing Characteristic Nodular Changes on the Exposed Surfaces

tuberculous lymphatic disease of the glands in both groins provides an obvious source of origin which the recorder appears to have entirely ignored.

Veit [4] describes a fibrous form and papillary form of overgrowth, and appears to consider that either form has its origin in lymphatic obstruction, the result of chronic lymphangitis. The inflammation is not dissociated from some septic or specific infection.

In my case there had been no source of irritation, no evidence of existing or pre-existing inflammation, and there had been no syphilis. The condition can only be explained by the constriction of the genitals produced by the forcible approximation of the thighs which had gradually increased as the spinal disease progressed.

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OBITUARY.

GEORGE HARRY MELVILLE DUNLOP, M.D., F.R.C.P.

It is sometimes very hard to realise that a friend is dead. That is the first thought that must have come into the minds of many of those who, on the 3rd of July, heard that Dr. Melville Dunlop had that morning died in France. Time had dealt so kindly with him that it seemed as though in his fight with sickness he would have had as his ally that youthfulness which had been one of his charms throughout his life.

George Harry Melville Dunlop was a son of the late James Usher Dunlop, and was born in 1859 in Leith, but spent his boyhood at Middlefield House, just across the Edinburgh boundary. He was educated first at Dr. Scott's well-known school in Picardy Place, and then at the Collegiate, at that time under the late Dr. Bryce, one of the principal Edinburgh day schools. There Dunlop did well, as the rows of prizes in his library testify. Medicine was not a tradition in his family; but to become a doctor was his natural bent, and he never thought of doing anything else. On leaving school he went straight to the University, and graduated M.B. in 1880, and took his M.D. in 1884. As a young graduate he acted as resident at Leith Hospital and at the Children's Hospital, and had a sea trip as ship's surgeon before he settled down to practice in Edinburgh, first in his mother's house at Middlefield, then in Abercromby Place. He was elected to the Visiting Staff of the Sick Children's Hospital in 1886, and became a Fellow of the College of Physicians in 1887. It would be difficult to say whether throughout the rest of his life his interest in the hospital or his pride in his College was the greater.

His chief work, of course, centred round the hospital. For a great part of his term of office as extra physician the indoor department was housed in temporary premises at Morningside, while the Dispensary was carried on at Lauriston Lane, and the attendance of students was small. When, on the retiral of Dr. James Carmichael in 1898, Dunlop was appointed full physician, the classes were becoming larger, and he had a chance of developing his powers as a clinical teacher. His cliniques were always popular and well attended: part of his success as a teacher resulted, I think, from the fact that he had singularly clear-cut pictures of disease, and possessed great talent for exposition, and part also from his power of putting himself in the student's place and realising what the men wanted to know. During the whole term of his office as physician he was constantly alive to the need

for change and reform if the hospital was to be kept abreast of the times, and he took a leading part in inaugurating improvements which would promote its usefulness to the sick. A reform for which all future generations of the staff have to thank him was the alteration of the term of service from ten to fifteen years—much needed change—which he succeeded in carrying through. He also, to a large extent, inspired among his colleagues that spirit of harmony and friendliness which does so much to oil the wheels of hospital routine. Outside his hospital work he was an examiner in medicine to the Royal College of Physicians and a physician to the Blind Asylum. Last spring he accepted an invitation to become physician to the Ministers' Daughters' College, work which, unhappily, he never lived to undertake.

Very near to his heart lay the College of Physicians, in all of whose activities he took the deepest interest, and of whose traditions he was very proud. The College appealed to him as a guardian of the highest standards of his profession and as a centre for friendly intercourse and dignified hospitality. He used to speak of the "loving care" which bygone generations had bestowed on its adornment, and just before the war he was much preoccupied by the idea of in some way enhancing the value and meaning of the Fellowship. For the past seven or eight years he had been a member of the council of the College.

As a physician Dr. Dunlop was a success from the first. His experience in early years was large and varied. The Leith Hospital of that time was very different from the present institution. Fevers of all kinds, especially typhus, were nursed in close proximity to medical and surgical cases; the out patient department was cramped, the patients many, and the staff small, so that the resident had plenty of work, and Dunlop made full use of his chances. He held, too, various clinical assistantships to the Infirmary, and although for many years he had restricted his practice to medicine, he used to tell with some gusto of his earlier operations on cases (parenthetically, for very small fees, too such as in later days he had to send to surgeons and specialists. His medical writings, though not very numerous, were universally recognised as valuable because they were based on his personal observations. He fully described one of the earliest cases of chloroma put on record in this country, and his paper on "Syphilitic Synovitis" was an important addition to our knowledge of the subject. He was one of the first to appreciate the importance of the thyroid treatment of myxoedema, as the date of his paper (1893) shows. He joined the staff of this *Journal* as a regular contributor at the time that the late Dr. George Gibson, one of his most intimate friends, became editor.

He had very good clinical judgment, and was seldom astray in diagnosis: in his treatment he trusted more to Nature, to good hygiene, to common-sense rules of life and diet than to drugs, and he was always profoundly surprised by the frame of mind which expects some

be-praised new remedy to work a miracle. He was sometimes a little worried by the way in which specialism nibbles at medicine, though his common sense showed him that such was inevitable and in the nature of things.

Dr. Dunlop's relationship to his patients was a singularly happy one. Something like fifteen years ago, when he was on holiday, one of them who came to consult the writer introduced himself by announcing —"I am one of Harry Dunlop's crowd." And so they all were. All spoke of him—many by his Christian name—with a mixture of affection and trust, which was enough to prove, even to one who did not then know him intimately, how exceptional a personality was his.

The qualities which always seemed to the writer to dominate Dunlop's character were his absolute straightforwardness in word and deed, and the vein of seriousness which underlay a light-hearted and buoyant manner. He was not only straightforward in his own dealings, but he was no respecter of persons; and if he thought a thing was wrong nothing would prevent his speaking his mind about it and trying to set it right. He was often consulted by his juniors, and he did not give advice heedlessly or casually, but seriously considered the circumstances and thought out carefully what had best be done. I think he used to be rather proud of the confidence thus displayed in his discretion, and certainly it was well deserved.

One could not be intimate with Dunlop for any length of time without realising the serious side of his personality; he was serious both because he was sincerely religious and because he had a curiously sensitive nature. Though he did not speak much about it, I know that he was greatly affected by the suffering and slaughter he was called upon to witness during this last year, and it was that probably more than any failing vigour which made him seem a little depressed when last he was at home. He was less interested in humanity at large than in his fellows as individuals, and the sight of so many cut off or crippled in their prime made poignant appeal to his sympathies. He was very charitable in his judgments of honest mistakes and stupidity, and was just as unsparing of what he felt to be wrong.

Of the more obvious side of his character—his high spirits, good nature, love of the fields and open-air sports, of good fellowship and the pleasures of life—it is unnecessary to speak. They gave to his personality the charm and geniality which made him friends wherever he went, as his more solid qualities enabled him to keep them.

An ardent Scot, Dr. Dunlop was a staunch supporter of the Church of Scotland, although he would never accept office therein. He leaves a widow and a son and daughter, to whom our thoughts now turn in respectful sympathy.

J. S. F.

Dr. G. H. Melville Dunbar

His Work in France.

To Harry Dunlop the call to duty was one that could not be stilled. He knew he had talents and experience that were not being fully utilised, and that would help his fellow-countrymen engaged in a life and death struggle abroad. No man had greater inducement to remain at home: a loving family, a host of friends, and a desirable practice; nothing could stop him, however: he must go.

His letter to the War Office must have been a convincing document, telling as it did, amongst other things, of experience of fevers gained in early years at the Leith Hospital, for within a few days he was offered the charge of the medical division of a general hospital about to leave on foreign service. Proud of this appointment, he showed a boyish enthusiasm in the making of preparations. The send-off from his native city, the few days' waiting at Aldershot, and the journey to France, seemed all part of a well-earned holiday, an illusion that was continued by the absence of work during the first six weeks on foreign soil. These were profitably employed in settling down to the life under canvas, and in becoming familiar with the complicated mechanism put together for the doctoring of a huge army. He threw himself into the new life with characteristic enthusiasm, with a lively appreciation of its many opportunities, so that when the serious work came along, as it did with a rush in the early days of August, he was amply prepared, and devoted himself to it with tireless energy. He was versed in the practice of medicine that cannot be learned from lectures or text-books, and for which there was a large demand. Because of his merits as a "doctor," but perhaps even more because of his personal charm and lovable character, the young-looking "major" became an institution in the camp, and his tent, of which he was almost childishly fond, became a centre of much that was human, and the Mecca of many who were in trouble.

He was much concerned in the medical problems raised by the war, but he did not take kindly to purely military regulations and observances. Always well turned out at home, the King's uniform showed off his figure to advantage. Between working hours he tramped the neighbouring hills, the seashore and the sand dunes, finding resemblances, but not requiring these to remind him of the Gullane to which he was ever devoted. He often recalled the days of "old Luffness," and the names of those who once frequented it. Keen golfer as he was, the close proximity to the camp of a well-known course did not attract him.

Part of the winter was spent with another unit at Versailles, but the resumption of town life did not appeal to him, and he gladly returned to the hospital by the sea. In the early spring, at the invitation of the Director-General, he made a tour round the front and

visited a number of old friends, including the writer. He appeared less youthful and less buoyant than heretofore, and not quite so pleased with things in general. He was keenly interested, however, and was much impressed with the desolation of Albert; the writer little thought when seeing him into the train at Amiens he was never to meet him in the flesh again.

His colleagues say he made a gallant effort to maintain the previous high standard of fitness, and probably he overdid the tramps on the hills and the bathing, especially after a hard morning's work in the wards. His spirit had always dominated his slim body, and he did not believe in giving in. On the 25th of June he was laid up with a pneumonia, which, with a pericardial involvement, brought about his end on the 3rd of July.

Although his death coincided with the height of the Allied offensive, when the energies of all were strained to the utmost, every officer in the district endeavoured to pay the last tribute to his memory.

He lies in the little cemetery by the sea which he had often visited in the course of his rambles, and no fitter resting-place could be found. Although unhallowed by the presence of the "forefathers of the hamlet," it is sacred for all time through the companionship of hundreds of other men who, like himself, had gloriously laid down their lives for their country.

A. T.

RECENT ADVANCES IN MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

W. T. RITCHIE, M.D., EDWIN MATTHEW, M.D., J. D. COMRIE, M.D., AND A. GOODALL, M.D.

The Spleen: Its Association with the Liver and its Relation to Certain Conditions of the Blood.

Wm. J. Mayo (*Journ. Amer. Med. Assoc.*, March 1916) discusses shortly the possible relationships between the spleen and the liver, and gives his results by splenectomy in the various forms of enlargement of the spleen. The spleen has a close and distinct relationship to the liver. It sends the material it takes from the blood to the liver; the spleen hypertrophies secondary to cirrhotic processes in the liver, and in Banti's disease (the terminal stage of splenic anæmia) the liver becomes cirrhosed. Probably the spleen conserves what is obtained from broken-down red cells, and sends them to the liver for further use, and also acts as a sieve for parasites and products of degeneration, and passes them on to the liver for conservation. The spleen also has a relationship to the blood. In the fœtus it is a blood-forming organ; at birth it loses power to produce red cells, and after birth it disposes of the worn out red cells. When the spleen becomes enlarged Mayo suggests this may be a work hypertrophy, as seen in splenic anæmia, hæmolytic jaundice, and possibly pernicious anæmia. In other cases the enlargement of the spleen—*e.g.* in syphilis—is definitely associated with the accompanying secondary anæmia, as is shown by the fact that removal of the spleen removes the anæmia. In enlarged spleens there are microscopic differences, and this, Mayo thinks, may explain why removal of the spleen is of benefit and may even cure conditions of the blood or liver which hitherto have been fatal. Mayo puts it that "the spleen—an organ not necessary to life and not causing disease on its own initiative—is the one link in an otherwise fatal chain which can be easily and safely broken."

Results of Splenectomy in Various Enlargements of the Spleen.—(a) *Infections.*—In primary tuberculosis of the spleen its removal has cured a few instances. In 3 cases Mayo removed very large spleens from cases of chronic syphilis with marked anæmia. Ordinary specific treatment had not the desired effect, but following splenectomy marvellous improvement in the anæmia took place. He removed the spleen in

7 cases of chronic recurring septic conditions. The benefits were not marked, such cases being liable to be carried off by cardio-renal insufficiency.

(b) *Splenic Enlargements in Association with Hepatic Disease.*—In primary biliary cirrhosis of young adults in which the spleen is also enlarged, splenectomy has been followed by marked benefit and even cure. In 4 cases of portal cirrhosis of the liver and greatly enlarged spleen Mayo removed the spleen with great improvement in 3 of the cases, the ascites and anæmia soon disappearing.

(c) *Enlargements in Blood Conditions.*—Cases of splenic anæmia are cured in most instances by removal of the spleen, and in several instances Mayo has obtained cure in the advanced stages of the condition, *i.e.* after the liver had become cirrhosed and ascites established. In 3 cases of Gaucher's disease in which the spleen was of enormous size Mayo performed splenectomy, with cure.

In hæmolytic jaundice—an anæmia of splenic origin with acholuric jaundice—Mayo has performed splenectomy in 9 cases, and with striking results. In 24 hours the jaundice begins to disappear, and in a few days the complexion is clear. The anæmia soon disappears, and the cure is permanent.

Mayo has removed an enlarged spleen in 19 patients with pernicious anæmia, and he is of the opinion that splenectomy, if performed in the early stages, will permanently check, if not cure, the condition.

Results from Blood Transfusion in the Treatment of Severe Post-Hæmorrhagic Anæmia and the Hæmorrhagic Diseases. Methods for Testing Donors for Transfusion of Blood.

Petersan (*Journ. Amer. Med. Assoc.*, April 1916) discusses blood transfusion and gives his results, and Minot (*Boston Med. and Surg. Journ.*, 4th May 1916) describes the methods for testing donors of blood before transfusion.

Petersan is inclined to think that at present the procedure is often adopted without discrimination. He believes transfusion ought to be employed in (1) cases of hæmorrhages of various degrees and types; (2) selected cases of anæmia; and (3) selected toxic and septic cases. Petersan's 27 recorded cases belonged to the first category of simple and pathologic hæmorrhage, the latter including hæmophilic and purpuric cases. In anæmia, from simple loss of blood—(*e.g.*) following trauma—transfusion of blood is always efficacious and prompt. In cases where the hæmorrhage cannot be reached without operation—(*e.g.*) gastric or duodenal ulcer or typhoid fever—transfusion does well, and, as a rule, it is better to wait till bleeding has ceased before transfusing. In chronic post hæmorrhagic anæmia following repeated losses of blood—

(e.g. hæmorrhoids, epistaxis—transfusion is easily the best remedy, and here it is better to employ several small transfusions than a single large one. All Petersan's 7 cases of this group are successful.

In hæmophilia, while the disease is not cured, transfusion offers the best results in helping those who have lost a quantity of blood. The transfused blood introduces into the patient an excess of the elements necessary for coagulation. Petersan's 6 cases were markedly benefited.

In purpuric conditions transfusion is of great service. He reports one case of hæmorrhagica neonatorum cured, and says that, from the experience of others, transfusion has reduced the death-rate from 60 per cent. to 5 or 10 per cent. In idiopathic purpura there is a tendency to uncontrollable hæmorrhage. Petersan treated 8 cases by transfusion. Two died, one of acute nephritis months after last transfusion, and one was moribund at the time the treatment was given. In 3 cases a single transfusion cured the condition. In 2 cases, where death was threatening from profound anæmia, transfusion was successful in saving them. In secondary hæmorrhagic diseases—(*e.g.*) in hæmorrhage seen in uterus, sepsis, nephritis, blood diseases, etc.—transfusion is useful in controlling the bleedings. In conclusion, he points out that transfusion may be a "miss or hit" game, because one donor's blood may do excellently while that of another has no effect, and that these variable results may be expected so long as we have no means of standardisation of blood as a therapeutic agent. Minot in his paper describes the essential conditions for successful transfusion. In transfusion he says it is desirable to have as a donor one who is healthy, whose blood gives a negative Wassermann, and whose plasma neither hæmolyses nor agglutinates the patient's red cells, and whose cells are not hæmolysed or agglutinated by the patient's plasma. It has now been established that "all human adults can be divided into 4 groups in regard to the ability of their red blood-cells to be agglutinated by other human sera, or their sera to agglutinate other human red cells." If, therefore, two individuals belong to the same group, neither will agglutinate nor hæmolyse each other's red cells. Some groups are less common than others; and the group to which a particular individual belongs is inherited, according to Mendel's law, and an individual throughout life, and in all conditions, remains in the same group. A donor is a satisfactory one if he belongs to the same group as the patient. At the Massachusetts General Hospital Minot has determined the groups to which a series of willing donors belong. When a transfusion case is admitted his group is determined, and a donor of the same group is summoned, and transfusion can then be done with no fear or risk of agglutination or hæmolysis. The determination of a particular donor's group is readily determined, and Minot, in his paper, describes the methods in regular use. Little apparatus is required, and the process of determining donors' groups takes only about 20 minutes.

a short-time method being essential, as transfusion is often required in an emergency.

The Pituitary Body and Renal Function.

Motzfeldt of Christiania (*Boston Med. and Surg. Journ.*, 4th May 1916) contributes further information on the relations of the pituitary gland to renal function. Clinical cases of diabetes insipidus treated with pituitary extract, and observations on normal individuals with pituitary, have led him to the conclusion that the pituitary gland controls the amount of urine passed and also certain of the solid constituents. In 3 cases with diabetes insipidus he was able to show that administration of extract of the posterior lobe of the pituitary gland considerably diminished the quantity and increased the concentration of the urine. In these cases, as röntgenograms showed, there was no tumour of the gland; and he concludes that his cases were the result of a hypofunctional activity of the hypophysis. This was corroborated also by the fact that they answered in their symptoms to the four cardinal symptoms laid down by Cushing as indicating a lowered functional activity of the posterior lobe of the pituitary. Motzfeldt is of the opinion that the majority of cases of diabetes insipidus are due to this lack in the posterior lobe. Following his clinical cases he made numerous observations on normal individuals, and ascertained that injection of extract of the posterior lobe had a distinct effect on the quantity of urine passed. In each case the diuresis was diminished, and in most cases to a considerable extent, and the concentration of the urine increased both as to nitrogen and sodium chloride. The effect of a single dose is not very lasting, and is not influenced by sex, age, or disease, and the results are obtained without any change in blood-pressure. This influence of the posterior lobe of the pituitary gland on the renal functions may be brought about in various ways—(1) Directly on the kidneys through the circulatory system; (2) through influences on the nervous system (autonomic or sympathetic); (3) through influence on other internal secreting organs. Assuming, Motzfeldt says, that all three factors are in action, the influence may be supposed to be exerted directly on the secreting epithelium of the kidneys or on the renal vessels.

Diagnosis, Symptomatology, and Therapy of Dilatation of Aneurysms of the Descending Thoracic Aorta.

Neuhof (*Amer. Journ. Med. Sci.*, May 1916) says that the object of his paper is to suggest that such aneurysms are of greater frequency than is supposed, that they have a definite symptomatology, and that the condition is a clinical entity. He describes 5 cases with

radiograms. The commonest symptoms are pains behind the sternum, or referred to different parts of the chest, neck, jaws, or head. Their etiology is not certain. Signs of heart decompensation are not marked. Œdema is slight and is confined to the legs. Dyspnœa is of the usual cardiac type. The left ventricle is hypertrophied to varying degrees. On auscultation abnormal sounds are heard at the left sternal border, at its middle third or from 3rd left intercostal space to the ensiform. There is a rough systolic murmur over the dilated aortal area. The second sound has a liquid rather than an accentuated tone, and is prolonged so as to occupy the whole diastole, or is followed by a diastolic murmur of varying intensity. A distinct heaving area, distinguishable from the apex beat, may be seen over the lower left intercostal spaces, and the thrill may also be felt. To complete the diagnosis fluoroscopy must be resorted to in several lateral positions. The prognosis in these cases, he thinks, is not grave, and as most cases are specific the treatment is clearly indicated. E. M.

SURGERY.

UNDER THE CHARGE OF

J. W. STRUTHERS, F.R.C.S., AND D. P. D. WILKIE, F.R.C.S.

The Treatment of Painful Neuritis by Injection of Alcohol.

Injection of alcohol into the trunk of a nerve has in the past mainly been employed in cases of severe trigeminal neuralgia, and with good results. In dealing with a mixed nerve, like the sciatic, it is important to realise that injection of alcohol into the nerve substance will cause not only anæsthesia in the distribution of the nerve, but also motor paralysis, which will last for some weeks or months. Hence, if this method of treatment is employed in cases of common sciatica, it is necessary to limit the injection to the neighbourhood of the nerve. A local reaction will be produced which may relieve the patient's symptoms and will not affect the motor nerve fibres.

Sicard (*La presse médicale*, 1st June 1916) states that he has employed alcohol by injection into the nerve trunks in cases of hemiplegia or paraplegia, in order to diminish spastic contractures. While improvement may occur in these cases it can only be for a limited period, as the lesion in the central nervous system is organic.

In the present war localised neuritis affecting nerve trunks is not uncommon after wounds by projectiles. It is difficult to explain why there should be so much more pain in some cases than in others. The nerve trunks most frequently involved are the median and

sciatic, more rarely the ulnar and anterior crural, and exceptionally the radial.

Reference is only made in this paper to very persistent neuralgias. In the majority of cases the painful symptoms associated with a localised neuritis, resulting from trauma, disappear in two or three months' time. In exceptional cases the pain persists and increases in severity, sometimes occurring in paroxysmal crises, so that the patient's condition is intolerable. The symptoms in such cases may closely resemble those experienced in trigeminal neuralgia; there is extreme hyperæsthesia to touch, and even a sudden sound may bring on a paroxysm which is sometimes associated with spasm of the muscles.

Treatment by the usual drugs and medical measures are of little avail in this type of case. Hot air, radiant heat, and vapour baths were tried in the author's series of cases, with other local measures, such as injection of gas into the subcutaneous tissues, injection of normal saline, etc., with little effect. Frequently repeated moist dressings gave most relief. Various surgical measures also were tried before adopting the use of alcohol injections, such as the freeing of the nerve from adhesions, or the injection of air, or of cocaine within the nerve sheath. In one specially severe case the patient suffered so severely as to threaten suicide, and was quite willing at any rate to lose his arm, if necessary, in order to get rid of the pain due to neuritis of the median and ulnar nerves. In this case the author divided the affected nerves above the lesion and then performed a primary suture in order to avoid permanent paralysis.

It is in intractable cases of neuralgia of this kind that the injection of alcohol into the nerve trunks is indicated.

Technique.—The nerve is exposed and freed from adhesions. A local anæsthetic is sufficient in simple cases, but if difficulties are to be met a general anæsthetic is indicated. No gross change may be visible in the affected segment of the nerve, or the latter may be thickened. The nerve should be exposed for a short distance proximal to the lesion, and 1 to 2 cubic centimetres of alcohol are then injected into the healthy nerve at a point 3 to 4 centimetres above the affected segment. The alcoholic solution should be from 60 to 80 per cent. in strength according to the chronicity of the case. Solutions of lesser strength than 60 per cent. are insufficient to alleviate pain. At the point of injection the trunk of the nerve becomes bulbous and pearly white in colour.

To ensure a successful result it is most important to inject the solution above the site of the lesion and in a part free from adhesions.

The author has so far treated twenty-one cases of neuralgia resulting from injuries received by soldiers in the present war, with most favourable results as regards the relief of pain.

The paralysis which is apt to follow is certainly a disadvantage, but when the aggravated nature of the cases is remembered the relief from

pain is well worth the disability produced by paralysis of the associated muscles.

Clinical and experimental evidence prove that motor paralysis, as a result of injection of alcohol, is only temporary. In the case of a motor nerve like the facial the paralysis of the facial muscles lasts from 8 to 10 months. Experimentally it has been shown that when 70 per cent. alcohol is injected into the nerve motor conductivity is interrupted from 6 to 10 months.

The histological changes which occur are degeneration of the myelin sheath and alterations in the axis cylinders. In the case of the latter the changes vary in proportion to the strength of the alcoholic solution—the axis cylinders may be hypertrophied or fragmented, but after a few weeks they gradually recover their normal appearance. The electrical reactions confirm the histological evidence as to the temporary nature of the changes. The reaction of degeneration may be complete after injection of 70 per cent. alcohol, but it tends soon to disappear, and after 4 to 8 months the electrical reactions are again normal.

Occasionally the injection of alcohol into a mixed nerve has a favourable effect upon the movements of the muscles supplied. Instead of paralysis following the injection the movements are improved. Presumably in these cases the motor nerve fibres have escaped damaged, and the improvement is due to the relief from pain due to the action of the alcohol on the sensory nerve fibres. Probably in these exceptional cases the paresis, present before the injection of alcohol, was due mainly to inhibition of the function of the motor nerve fibres as a result of painful stimuli.

The Final Results of Colectomy.

Clark (*Surg., Gynec., and Obstet.*, May 1916) records the results of excision of the colon in a series of twelve cases operated upon for the relief of chronic constipation and the associated symptoms of intestinal toxæmia. On a former occasion the author had published the facts of some of the cases; but, as certain of those which had then been reported on favourably had since failed to maintain the improvement, it was thought advisable to publish the final results of all the cases.

The technique employed in most of the cases was excision of the colon and a lateral anastomosis between the terminal portion of the ileum and the sigmoid. It should be noted that the modifications in the technique introduced by Lane had not been used in this series of cases. Lane at first used a lateral anastomosis between the ileum and sigmoid; later he recommended a lateral implantation of the lower end of the ileum into the pelvic colon; more recently still he has abandoned these methods in favour of an end-to-end junction between the ileum and sigmoid, the rest of the colon having been excised.

The author has practised a partial resection of the colon in one or two of the cases, the ileum being joined to the transverse colon.

In following the subsequent progress of the cases the author's enthusiasm has diminished rather than increased. In the series of twelve cases there was no primary surgical death, but one patient died four months later from acute obstruction, due to strangulation by a band, in spite of a second operation. A second operation was required in two other cases.

In all of the cases there was a great improvement in the constipation for a time. In six of the cases the results were entirely satisfactory, there being great improvement in nutrition as well as relief from constipation. There was no tendency to prolonged diarrhœa, and any excessive looseness of the bowels was easily controlled by simple medicinal means.

In the remaining six cases there was no improvement in general health, and in four of the cases there was a gradual return of severe constipation; in three of the latter cases an X-ray examination showed that the lower end of the ileum was dilated to a size closely resembling that of the colon.

As a result of his experience Clark is of the opinion that total removal of the colon is justifiable only in severe cases of obstructive constipation. He is inclined further to recommend a less radical operation and to limit the colectomy to the cæcum, ascending colon, and part of the transverse colon, completing the operation by a lateral anastomosis between the ileum and transverse colon. By this method the omentum is saved, and there is less traumatism and subsequent risk of adhesions. The disappointing results sometimes met with, after an apparently marked initial improvement following colectomy, are due either to post-operative adhesions or to the effects of backward pressure from the colon through the anastomotic opening causing dilatation of the ileum and impairment of its function.

The Treatment of Chronic Empyema.

Robinson (*Surg., Gynec., and Obstet.*, May 1916) states that no one method of operating on chronic cases of empyema is likely to be uniformly successful.

A combination of the chief features of several or of all the types of surgical treatment most often effects a cure. No operation of dangerous magnitude should be undertaken; the surgeon should rather operate in stages, closing each stage at the moment which is consistent with the patient's recovery. A large lateral empyema will thus take 12 or 14 months before being cured and require, possibly in the first place, decortication; later, after a period of three months or so, removal of ribs, as in Estlander's operation; again, three months later, a Schede

operation, possibly modified by implantation of muscle: finally, a small cavity may remain, which can be successfully obliterated by injections of bismuth paste.

After making a diagnosis the size of the cavity should be ascertained. This may be done by introducing a flexible olive-tipped probe, which is moved and rotated in all directions. The most exact method is to pack into the cavity with a probe a narrow bandage or tape, which has been impregnated with barium sulphate mixed with water to the consistency of a cream. A stereoscopic radiogram is then taken, and the shadow formed by the tape will be readily distinguishable from the lung shadow even when the two are superimposed.

The injection of a liquid mixture of barium or bismuth is not so good, as it is difficult to completely fill the cavity, and, if the sinus is small, most of it may be retained and cause a reaction.

No operation should be performed for obliteration of a chronic cavity until the cavity has been drained freely at its lowest limit for a period of six weeks. Any openings which are already present are usually too high, and it is advisable to resect a portion of rib, after demonstrating the bottom of the cavity by probe or needle: after a few weeks' efficient drainage the patient's condition should be much improved, and he should be able to stand further operation if necessary.

The desired objects of the operation are either to cause a sinking in of the chest wall, or to fill up the cavity by encouraging expansion of the lung by decortication, or by implantation of other tissues. Obliteration of the cavity is the main difficulty even when there is an extensive resection of the chest wall, as in Schede's operation. There is always a tendency for the skin and muscle flap to bridge the cavity rather than to adapt itself in its contour.

To avoid this difficulty Robinson has modified Schede's operation by implanting muscle flaps into the cavity, which is thus partly filled up.

The first step in the operation is the reflection of a large U shaped flap of skin and muscle; the portions of ribs overlying the cavity are next resected subperiosteally, and the intercostal vessels and muscles ligated. The thickened parietal layer of pleura is removed. A muscle flap is now prepared by splitting the latissimus dorsi which forms part of the flap raised from the chest wall. One half of the muscle is separated from the overlying skin and sutured into the upper part of the cavity; the remaining half is implanted in a similar way. If now the skin flap were stitched in place it might bridge the cavity as a tight membrane, leaving a space between it and the implanted muscle. To avoid this the skin flap is split vertically and a triangular portion of the flap is removed from the curved edge. The U shaped flap is thus converted into two lateral ones which can be kept in contact with the muscle, leaving a triangular gap below which is convenient for drainage.

The author has also employed an inverted T-shaped incision, forming two lateral flaps which are laid over the resected ends of the ribs, the edges of the flaps being stitched to the depths of the cavity.

J. M. G.

DISEASES OF CHILDREN.

UNDER THE CHARGE OF

W. E. DRUMMOND, M.D., AND A. DINGWALL FORDYCE, M.D.

Dyspituitarism in a Girl of Fifteen.

A case of this rare condition is described by Stephenson in the *British Journal of Children's Diseases* (May 1916). The chief points in the case were—(1) The existence of bilateral simple optic atrophy, otherwise unexplained; (2) loss of the temporal field in the left eye (temporal hemianopsia); (3) X-ray evidence of enlargement of the sella turcica; and (4) the general appearance of the girl, who was inclined to stoutness and presented slight prominence of the eyes and slight strabismus. Menstruation had not begun.

The Problem of Mental Deficiency.

Sajous contributes to the *New York Medical Journal* (1st April 1916) a long paper on "Our Duty to Mental Defectives of the Present Generation." He considers that eugenic measures, such as segregation, are too slow in their operation, while any legislative impediments to marriage defeat their own ends by their tendency to promote immorality. A very grave view of the problem is taken by the author, who says he cannot but agree with Dr. Kehoe when he states that the time is not far off when "to see an individual of natural poise, normal mind, and healthy body will be the exception and not the rule."

In spite of this exaggerated estimate of a very real social danger, Sajous is not without hope and belief that the problem may be solved. The prophylactic measure he proposes is the extensive resort to organotherapy, not only in the treatment of mentally defective infants, but—with greater probability of success—in the treatment of their mothers during pregnancy. The strongly hereditary character of mental deficiency he seeks to explain as the result, not of the absence of some Mendelian factor, but of a deficiency in the secretion of some ductless gland. He asserts that we have in the functional disorders of these

organs the underlying cause of several of the most common mental diseases, and that it is because these glandular disorders are virtually overlooked in the pathogenesis of many psychoses that the painstaking efforts of innumerable psychiatrists during the last sixty years have remained so sterile of results.

The author then proceeds to give a *résumé* of the association between disorders of the ductless glands and mental symptoms. Thus in Addison's disease, in which organic lesions of the adrenals predominate, we may have melancholia and, when the auto-toxæmia is marked, mania. Again, in deficiency of the thyroid apparatus, two striking mental disorders—melancholia and mania—may be witnessed in addition to the idiocy of myxœdema and, in children, cretinism.

Disorders of the pituitary also give rise to psychoses. Here, again, we meet psychasthenic states of a melancholic type sometimes attended by delusions of persecution, convulsions, etc., all due to defective destruction of tissue wastes.

The thymus is so important an organ in this connection that it may be said to stand, in respect to idiocy and dementia præcox, as the thyroid does to myxœdema and cretinism. It is the gland upon which the brain-cells depend for their development supply of phosphorus laden nucleins; if it fails, through organic disease, defective development, or premature involution, to furnish its product, the organ of mind remains undeveloped, and idiocy results. Dementia præcox the author regards as due primarily to inhibition of the thymus too early at puberty, *viz.* before the completion of the development of the brain. Disease of the pineal gland has been associated with remarkable precocity of intellect.

To sum up, any disease capable of injuring the ductless glands sufficiently to inhibit their functional activity impairs correspondingly the development and functional activity of the brain, by reducing the supply of secretions this organ requires to carry on these physiological processes. The evidence is sufficient to justify the assertion that the main underlying cause of defective mentality in both parent and offspring is inherited deficient activity of the ductless glands. Under these circumstances it is evident that we ought to start a campaign, having in view the salvation of these unfortunate infants, by supplying, through the intermediary of their defective mothers, and, after birth, through their food, the secretions they lack to complete their development. This could be accomplished by administering organic products to the mother during pregnancy and while she is nursing her infant, or if, as is the rule, the latter is fed artificially by the addition of the organic products to the cow's or goat's milk used. To determine the course of treatment indicated the main stigmata of deficiency of each of the major ductless glands should be ascertained. These main signs are as follows:—

Stigmata of Thyroid Deficiency.—Subnormal temperature, a doughy, dry skin, mental torpor, and other well-known symptoms.

Stigmata of Thymus Deficiency.—(1) Deficient development of the osseous system, with defective development of epiphyses and deformities suggesting rickets or osteomalacia; undersized stature. (2) Deficient mental development. (3) A low relative lymphocyte count due to the inadequate formation of these cells by the thymus.

Stigmata of Adrenal Deficiency.—(1) Muscular weakness and emaciation, pallor, deficient hair growth, sensitiveness to cold, subnormal temperature, all due to deficient tissue oxidation and recession of the blood mass into the splanchnic area. (2) Weak heart action and pulse, low blood pressure, and constipation due to deficient peristalsis, the result in turn of torpor of the intestinal muscular layer. (3) Pigmentation, sometimes limited to bronze areas on the back of the hands, and freckles. (4) Mental torpor, slow intellection or even idiocy where the adrenal deficiency is initiated *in utero*.

Stigmata of Pituitary Deficiency.—(1) Subnormal temperature, low blood-pressure, pigmentation (symptoms of deficient activity of the adrenal medulla), muscular weakness and scanty hair growth; also in a pre-adolescent case, undeveloped or infantile genital organs (symptoms of deficient activity of the adrenal cortex). (2) Tendency to adiposis; smoothness, but in some cases roughness and dryness, of the skin (symptoms of deficient activity of the thyroid). (3) Undersized growth owing to deficient skeletal development and imperfect ossification of the epiphyses (symptoms in a pre-adolescent case of deficient activity of the thymus).

Sajous considers that a great deal of mental deficiency could be prevented by timely treatment of pregnant mothers according to the stigmata present, but he admits that in numerous cases no clearly definable stigmata are recognisable in the parents. By a rather remarkable jump, he concludes that this means that any infant may become a defective unless its development is closely watched by a physician. A large proportion of defective children might be cured by early treatment with organic products, combined with attention to the special senses. Obstetricians should watch the kidneys carefully during pregnancy, as the accumulation of toxic substances may injure the ductless glands of the fœtus. In early childhood the infectious fevers are a source of danger to the ductless glands, hæmorrhage into which may compromise their functions sufficiently to arrest cerebral development.

Mental deficiency is not the only condition which might be diminished in pregnancy by organo-therapy. Twenty thousand cases of dementia præcox are committed to insane asylums each year. If all general practitioners familiarised themselves with the early symptoms of these cases, they could, by means of organo-therapy,

arrest the morbid process in at least a large proportion of them.

Syphilis and Mental Deficiency.

Many authorities, such as Mott, hold that syphilis is a factor in the causation of mental deficiency in many cases which are free from all visible signs of that disease. The results obtained by the Wassermann reaction, however, differ so greatly that but little reliance can be placed upon them. Shuttleworth and Potts, in the new edition of their well-known text-book, give a table showing that the positive reactions obtained by different investigators range from 1·5 per cent. to 60 per cent. One of the most recent papers is by Goodall and Scholberg (*Lancet*, 1915, vol. ii. p. 1457), who found only 6 per cent. of positive Wassermann reactions in epileptics, and 3 per cent. in non-epileptic defectives. However, they report on only 46 cases.

W. B. D.

DERMATOLOGY.

UNDER THE CHARGE OF

F. GARDINER, F.R.C.S., AND R. CRANSTON LOW, M.B., F.R.C.P.

The Vaccine Treatment of Certain Chronic Pyogenic Dermatoses.

Dunnie and Bufford (*Boston Med. and Surg. Journ.*, December 1915) record the result of a series of skin cases treated with vaccines. All the cases were chronic, the disease having lasted for more than three months. Most of the cases had had several kinds of local treatment without benefit. No external treatment was used while the vaccines were being given. Some cases were treated with stock vaccines and others by autogenous vaccines, and the results compared. The stock vaccines used were prepared by the authors, and consisted of excess preparations of the autogenous vaccines. The initial dose was not less than 100 million, and the last injection was often 2000 million. The administration was governed by the local reaction at the point of injection, such as redness, heat, slight oedema, and tenderness on pressure. These symptoms appeared within from four to twelve hours after administration, and sometimes lasted as long as forty-eight hours. If this reaction did not occur after one of the early injections a new preparation was made and administered. If no reaction occurred the authors concluded either that the right organism had not been obtained

in culture or that its properties had been injured in the process of preparation. The general reaction was usually so slight as to be imperceptible to the patient. When there was an increase in the intensity or number of eruptive elements the following dose was much smaller and only given when the untoward symptoms had disappeared. If the patient improved up to a certain point and then remained at a standstill a new vaccine was prepared, as the authors consider that in such a case either the old preparation has lost its potency or the organism has built up an immunity to its own toxines. If the patient grew progressively worse after each injection the treatment was discontinued. During the vaccine treatment the acne patients were placed on a diet from which milk, eggs, butter, potatoes, bananas, pastries, soups, and all highly seasoned foods were excluded. The diet was regulated so as to correct constipation and to prevent overeating.

Seventy cases were treated as follows: Acne vulgaris, 35 cases; furunculosis, 21 cases; folliculitis (including sycosis), 14 cases. Sixty-four per cent. of all cases treated were apparently cured, 20 per cent. were benefited, and 16 per cent. received no benefit whatever. Autogenous vaccines were superior to stock, excepting in the treatment of furunculosis, where no difference could be seen. In most cases a permanent cure resulted from the use of the vaccines. There were only two recurrences in those who reported at the end of one year. In acne the best results were obtained in the indurated type. The comedo type responded less favourably, and the furuncular type least favourably. Of the three pyogenic dermatoses the best results were secured in furunculosis. The action of the vaccines in sycosis was very erratic. In one case of twenty years' duration, where external treatment had been used for five years, and where stock vaccines had been used, the autogenous vaccine was remarkably successful. On the other hand, two cases were very much aggravated by this form of treatment. The average number of doses administered in successful cases was eight in acne, four in furunculosis, and four in sycosis. The most favourable interval between doses was found to be five days in acne, four days in furunculosis, and seven days in sycosis.

Dermatoses Developed around Wounds and Fistulous Openings.

As inflammation of the skin around wounds and fistulæ are very common in soldiers at the present time it may be useful to give the experience of Major Desaux (*La presse médicale*, 30th March 1916). These dermatoses may be divided into two groups:—

1. Those of microbic origin. These are inflammations of the dermis

and epidermis produced by the organisms which develop on the tissues injured by antiseptics.

Those which are local manifestations of general cutaneous eruptions in persons previously predisposed or affected. The two types of course may be combined in the same case. The wound is usually situated on a limb. The projectile which has produced the wound has broken the bone and the fracture has suppurated. When the skin lesions appear (three, five, or even ten months after the wound) the old wound has become the orifice of a fistula which communicates with the infected bone. An abundant yellowish pus escapes from the opening, and the pus contains abundant streptococci. It is surrounded with a ring of scar tissue covered with a very thin epidermis, reddish violet in colour, partly anæsthetic, and often adherent to the deeper tissues. It is a tissue which is easily injured. The surrounding skin is in a state of lowered resistance. In some cases, like the whole skin of the patient, it is very dry or sensitive to external irritants; in other cases its feeble vitality is the result of local traumatism by the projectile. The vascularity is poor, the skin becoming readily cyanosed and cold; the nerves have been injured, and cutaneous sensibility may have disappeared. The bony callus compresses the adjacent tissues and interferes with the nutrition of the skin. The muscles, if destroyed, are replaced by fibrous tissue, which, by its contraction, pulls on the skin; and, lastly, numerous radiographs have been taken, so that it is possible that the X rays so applied have a deleterious effect on the skin and blood vessels. The antiseptics used for the fistula have undoubtedly a bad effect, especially the tincture of iodine, which is often applied daily for long periods. The wound and its surrounding skin soon become itchy. This itch usually precedes the skin lesions by some days, and should serve as a warning what to expect. The epidermis of the scar pales, rises up, and comes off on the dressings. The surface is now raw and oozes with a serous discharge. On to this raw surface the pus containing streptococci from the fistula is poured out. The devitalised skin is at once infected, and in spite of stopping the use of the iodine or other antiseptic, or in spite of the healing of the fistula and the drying up of its discharge, the dermatitis spreads to the surrounding skin. As the lesions spread the edge is usually very itchy. The dermatitis usually spreads out with a fairly sharp, often festooned, edge into a large roundish or oval patch. Sometimes the patch is covered with thick yellowish, soft, often stinking, crusts under which pus collects.

After removing the crusts the surface is red, moist, and "weeping." It is the typical picture of impetigo. Later the surface becomes "varnished" with a very fine transparent epidermic membrane, which is ruptured in some places. From these openings an abundant serous discharge pours out. In this thin skin pustules or hæmorrhages may

appear. Last of all, the patch may look rather like one of psoriasis covered with whitish, dry, transparent, slightly adherent scales. Underneath this scaly covering is the thin red membranous skin. Any slight injury causes this dry scaly lesion to become moist again. Around the large patch in the healthy skin other lesions appear, and so the disease spreads.

Desaux found that the pus of the fistula gives almost a pure culture of streptococcus. Exceptionally this organism is accompanied by the staphylococcus. In the fresh spreading skin lesion the streptococcus alone was found to be present. Later, in the serous fluid and scales, the staphylococcus was found to be abundant. In the pus from the fistula other organisms were occasionally found, especially two forms of bacilli, which seemed to be saprophytic.

Treatment of such cases should be in the first place preventive. Iodine or other antiseptics should not be used after the skin around the wound begins to get itchy. The author recommends a simple zinc paste dressing for the skin around the fistula as the best preventative. When the dermatitis is developed the crusts should be softened and removed, and the surface touched with 1 per cent. silver nitrate solution and dressed with a 10 per cent. collargol ointment. At the same time, of course, the fistula is treated surgically, taking great care that no antiseptics are allowed to drop on to the surrounding skin. After some days, when the surface becomes red, shining, and oozing, it should be treated with a paste containing a little tar and ichthyol. Later, as the surface dries up, stronger tar may be painted on occasionally. On the slightest irritation the surface is apt to become moist or slightly itchy and scaly at places. These are not infected lesions but traumatic, due to the lowered vitality of the tissues or to trophic causes. The best treatment in that stage is the continued use of tar and gentle massage to stimulate healing.

In the second group of cases there is usually a previous history of skin trouble, either eczema or psoriasis. Prolonged stays at the front, and lowered general vitality, tend to bring these conditions into prominence. The wound still further lowers the local vitality, and naturally the disease appears very readily on the injured area. Occasionally the skin lesion develops generally, and for a long time does not affect the area of dermatitis around the wound. The treatment used should be that suitable for the skin condition which is superadded.

Secondary Lichenoid Trichophytides in Association with Ringworm.

Rasch (*Bull. Journ. Dermat.*, No. 1, 1916) reports two cases of this curious condition. Both cases showed kerion ringworm of the scalp,

and both showed on the body an extensive rash which resembled lichen spinulosus. This condition was first described by Jadassohn in 1911. Jadassohn describes it as occurring frequently in association with kerion of the scalp, especially about the time of healing. The eruption consists of small pale follicular papules, sometimes very numerous, which after some time disappear. They most closely resemble lichen scrofulosorum, but do not react to tuberculin injections, and are also histologically different from that disease. Occasionally they show small horny spikes or spines, and then the condition corresponds to the picture of lichen spinulosus. He believes these eruptions, associated with deep seated tinea, are exogenous auto-intoxications. Other observers have seen the condition fairly frequently in kerion of the scalp in children and deep-seated ringworm of the beard in adults. Guth even goes the length of stating that all children with kerion show the condition to a greater or less degree. The rash is usually unaccompanied by subjective sensations. It may either appear suddenly at one time, or may extend by repeated outbreaks. It varies greatly in its extent, duration, and appearance, being found most extensively on the body, to a lesser extent on the limbs, and only in a few cases on the face. The lesions may be diffusely spread or be grouped. Sometimes they disappear in a few days, but they may last for several weeks. In the majority of cases the eruption consists of miliary follicular, pale or pink, pointed or flat, papules, which sometimes have on the summit a small scale or crust. More rarely small vesicles or pustules are present. When about to disappear they develop a quite small scale. In some cases they may develop white bristles or spines, so that the condition is sometimes called lichen spinulosus trichophyticus. Microscopically, the condition shows a follicular and perifollicular inflammation. In the spinous form there is considerable parakeratosis in the wall of the follicle, and the horny layers are pushed outwards by the pressure inside, and so give rise to the projecting spine.

The diagnosis has always to be made from lichen scrofulosorum. This latter disease is, as a rule, more limited, more grouped, and of a darker bluish red colour. The evolution is slower, and there is much less tendency to develop spines. Of course the associated kerion or tuberculosis lesion helps in the differentiation. With regard to lichen spinulosus, it is doubtful whether some or all of the cases which have from time to time been published under that name are not lichenoid trichophytides. The affection may also closely resemble pityriasis rubra pilaris; but in children the latter disease usually affects the palms, and soles, and face, and is of a much more chronic nature. As to the origin of lichenoid trichophytides, they might be due to multiple exogenous inoculation, or to a hematogenous infection with fungus or its toxines. There is no evidence to show that it is of exogenous origin. No fungus

is to be found in the lesions (only Guth has, on one occasion, found a single mycelial thread). Its general exanthematic appearance points to some internal cause, either by the absorption of a toxine or particles of dead fungus from the kerion lesion.

R. C. L.

NEW BOOKS.

On Modern Methods of Treating Fractures. By ERNEST W. HEY GROVES. Pp. xii. + 286. With 136 Illustrations. Bristol: John Wright & Sons, Ltd. 1916. Price 6s. 6d. net.

MR. HEY GROVES' work on the subject of fractures is well known, and the publication of this summarised expression of his opinions and practice is most timely. While belonging to the more advanced school of thought with regard to the treatment of fractures, Mr. Hey Groves is in no way biassed, and he fully recognises, and, indeed, one of the objects of his book is to emphasise, that no one method can be followed to the exclusion of others. Mobilisation, massage, extension, and operative treatment are all at our disposal, and each plan has its appropriate occasion, and the selection of one or other method or combination of methods is determined by the requirements of the case and not by any preconceived ideas as to the superiority of one plan over all others. These various methods are not to be regarded as "independent, rival, or mutually distinctive systems." Throughout the work the author rightly insists that the problem before the surgeon in the treatment of fractures is largely a mechanical one. In the introductory chapter this matter is fully discussed, and the principles on which treatment is based are clearly and convincingly enunciated.

The chapter on massage and mobilisation gives a fair and reasoned statement of the present-day views on the plan which Lucas Championnière did so much to popularise. The warning that the word "massage" in this connection connotes something totally different from the "manifold pressures and kneadings" of the professional masseur should be taken to heart by all who employ this treatment for fractures. "A gentle superficial stroking, a soothing caress" is the author's direction to the rubber, and "the treatment by hypnotism" is the name he suggests for the process. This seems to us to sum up the position.

On the diagnostic use of the X-rays the author is somewhat dogmatic, and he inclines to condemn the writers of text-books for describing the various manipulations necessary for diagnosis when a radiogram will give so much more accurate and full information. We fear that until an X-ray apparatus is available in every town and village in the land it will be necessary for teachers of surgery to instruct their students how to diagnose a fracture without the aid of the X-rays. It is no longer necessary to emphasise the inestimable value of radiographic evidence, but it is often not possible to obtain it.

The sections devoted to the extension methods are of special value at the present time when so many cases of compound fractures are coming under notice. The various ingenious and useful (we speak from experience) splints devised by the author are described and illustrated.

The greater part of the volume is devoted to the consideration of operative treatment, based upon experimental and clinical observation. The various methods of securing the fragments are clearly described, and the procedures are well illustrated.

We can confidently recommend this work to our readers.

The Drink Problem of To-day in its Medico-Sociological Aspects. Edited by T. N. Kelynack, M.D. Pp. xii. + 318. London: Methuen & Co., Ltd. 1916. Price 7s. 6d. net.

The publication of this book is very opportune, seeing that danger has stimulated the national conscience and has revealed weak points in our national life in its relation to alcoholics. Russia, France, Austria, Germany, Italy, and Switzerland have all taken steps to raise the efficiency of both the military and the civil population by increasing the control of the sale of alcoholics. In a speech which he made in 1910 the Kaiser expressed the opinion that in the next war the nation which drinks the least alcohol will be the winner.

The volume is almost entirely a new work. It consists of fourteen articles on various aspects of alcoholism—its biology, pathology, psychology; its relation to crime, poverty, work, war; its influence on women and children, etc. These are written by well-known experts in their special subjects. The editor—well known for his work in relation to "Inebriety"—contributes an article on "The Arrest of Alcoholism." The various subjects are treated from a scientific standpoint, in a broad, fair-minded spirit; each has its own special character and interest. They are nearly all of special value to the physician and surgeon. The article on "Alcohol and Life Assurance" presents very many important points and reveals a most astonishing change by life assurance companies in their attitude to total abstainers. That on "National Efficiency" records the illuminating lesson of Russia's self-discovery because of the total prohibition of vodka. It records the immediate results in national savings and amount of work, also the less immediate effect on pauperism, vagrancy, crime, lunacy, disease, and premature death. It is interesting to find recorded in "The Development of National Sobriety" that the genesis of temperance education in its scientific sense is traced to a "Remonstrance against the Common Sale of Ardent Spirits," which was made by the Royal College of Physicians in 1725. The article on "Legislation" strongly

advocates the establishment of free clubs, warm, well lighted, where men may smoke, read, play games, meet friends, with no intoxicants, no direct moral teaching, nor lectures.

The Pathology of Tumours. By E. H. KETTLE, M.D., B.S.(Lond.), Assistant-Lecturer on Pathology, St. Mary's Hospital Medical School. Pp. viii. + 224. With 126 Illustrations. London: Lewis & Co. 1916. Price 10s. 6d. net.

IN this volume the author deals with the general and special pathology of neoplasms. He divides the subject into three parts. The first treats of the general biology of tumour growth, etiology, modes of growth, gives a good account of the experimental study of cancer, and includes also a chapter on treatment, though we are surprised, in a special consideration of the subject such as this is, to find no mention of the autolysate treatment of cancer, so ably advocated by Fichera and others. The second part deals with the general pathology of tumours, methods of classification, and the general characters of the different types of tumours. In the third part the special pathology of tumours, in their relationship with the different tissues and organs in which they occur, is considered. Perhaps the best chapters in the book are those on the experimental study of cancer, on endotheliomata, and on hypernephromata. There are few statements to which exception can be taken, but we must admit a long standing prejudice against such terms as carcinoma *simplex*. The illustrations are numerous and well chosen. The drawings of microscopical appearances are almost without exception excellent, and form an important and valuable feature of the book. The photographic representations of naked-eye appearances are not so satisfactory.

The book can be recommended to students and to practitioners who wish to obtain information about tumours of a more advanced standard than is usually found in good general text-books on pathology.

The Cure of Obesity and Other Ills. By J. S. KELLETT SMITH, F.R.C.S. Pp. 93. With 12 Illustrations. London: J. & A. Churchill. 1916. Price 3s. 6d. net.

THE writer draws attention to the value in the treatment of obesity by the method of Professor Bergonie, who in 1909 recommended for this condition the production of muscular contraction by the faradic current. The method called "Passive Ergotherapy" consists in the application to the muscle groups of the limbs and abdomen of a faradic current rhythmically interrupted and reversed 120 times per minute.

By this means the main muscle groups are thrown into energetic rhythmical contraction, any disagreeable stimulation of the skin being avoided by the character of the current employed and by its application to a large part of the body surface. Sandbags placed upon the abdomen and limbs serve to preserve contact of the electrodes during the muscular movements, to limit the extent of muscular shortening, and to increase the work done by the muscles during treatment. Electrical and other details of the apparatus employed are given. Muscular exercise is obtained in this way without any expenditure of energy on the patient's part in initiating and controlling the movements. As the aim of the method is to increase tissue loss, diet must be supervised, fluids, fats, and especially carbohydrates being reduced. In addition, the patient is encouraged to take voluntary exercise, which may with advantage take the form of regulated ascent of various gradients on Oertel's plan. The muscular contractions induced by the current greatly assist the venous return, and as the peripheral arteries are at the same time opened up, the circulation is materially improved in the frequent cases of obesity in which fatty deposits are hampering the work of the heart. The claims advanced for the method are supported by citation of various types of cases which benefited from its use, and the impression given is that the method offers distinct promise of aid in the treatment of what is often a very troublesome and baffling condition.

Studies in Surgical Pathological Physiology from the Laboratory of Surgical Research, New York University. Edited by J. W. Draper, M.D. Vol. I. Pp. 234. With 141 Illustrations. New York: University Publisher. 1915.

This is a volume which includes reprints of the original articles contributed by a number of workers in the Surgical Research Laboratory of New York University to various American journals. These all appear in the form in which they were originally printed, with the result that the pages present a very heterogeneous collection of type and paper which is not at all pleasing. The term "Surgical Pathological Physiology" impresses one as being at least an unusual one.

The subject matter is, however, of an extremely interesting and valuable nature. In a short introduction the editor emphasises the importance of physiology in determining successful surgical procedure, and is very emphatic in his denunciation of the purely "technical" surgeon. W. J. Mayo, who contributes a foreword, notes what is certainly the most valuable feature of the book, viz. the collection into one volume of a number of articles bearing on similar lines of research, which should prove very helpful to others engaged in considering the same problems. There is a large bibliography.

In the first article, "Medicine and the Great War," in the course of a plea for a more thorough and scientific training for the American surgeon, the editor remarks, "For many years, if not permanently, the devastation of Europe will place progressive medicine in the hands of America."

There are seven very interesting papers, written chiefly by Draper and Lynch, on the physiology of the alimentary canal, with special reference to the large bowel. These contain a summary of the results of experiments made by them upon dogs and also a number of observations in a case of multiple polyposis in a physician treated by them by ileostomy. Some of the conclusions arrived at are at variance with those at present generally held, and many of the facts are new. The suggestions made as to the part played by the epithelium of the small intestine in neutralising toxins and its relation to the toxæmia of acute obstruction are, if correct, worthy of consideration in the treatment of this condition. But the greater part of this portion of the book is devoted to "ileal stasis" and the operative treatment of this and other forms of constipation, special attention being directed to the operation of ileostomy and the indication for this operation. In general, the conclusions are not dogmatic, but the facts which are brought out are very illuminating and should stimulate further research.

Sharpe contributes an excellent article on "Spina Bifida" and its relation to intradural pressure, together with an account of its varieties, symptoms, diagnosis, and operative treatment.

Hydronephrosis and renal infection are discussed by Stewart and Barber from the standpoint of the effect of paralysis of the ureter, on the spread of infection, and on the function of the kidney.

Janeway and Ewing report some experiments bearing on the cause and production of shock, and conclude that paralysis of the splanchnic area is a very important factor.

A number of technical papers completes a most enjoyable volume, of which Mayo says, "Its contents are representative not only of the surgery of to-day but of to-morrow."

Localisation by X-Rays and Skiascopy. By Sir James Mackenzie Davidson, M.B., C.M. Pp. xi. + 72. With 26 Illustrations. H. K. Lewis & Co., Ltd. 1916. Price 7s. 6d. net.

It is unfortunately the case that, even at this late period, accurate localisation by means of the X-ray is neither thoroughly understood nor universally practised. Sir James Mackenzie Davidson's admirable little work recently published should go far to stimulate effort in the direction of more precise determination of the position of foreign bodies in the tissues.

The parallactic displacement is undoubtedly the only accurate basis from which to compute these positions, and the Mackenzie Davidson localiser still remains a standard instrument whereby these positions are determined.

In practice, radiograms are usually taken at distances of 50 or 60 centimetres, with a tube displacement of 6 or 10 centimetres, and with these it becomes easy to construct a graph, which, without the aid of a localising apparatus, further facilitates the localisation of foreign bodies. To this advance in method the author has unfortunately not adverted. An admirable chapter (VII.) is devoted to the localisation of foreign bodies in the eyeball and the orbit, and the method therein demonstrates how easy it is to obtain accurate results by a simple process.

Any pair of photographs taken for determination by parallax can be used in the stereoscope, and the recommendation of the employment of this instrument cannot be too strongly emphasised. Other instruments less costly and cumbersome than the Wheatstone stereoscope give equally good results. With the Pirrie (British) and the McAllister (American) stereoscopes it is easy to observe stereoscopically any pair of plates, and the cost of either of these instruments is under £3, while their bulk is not larger than a pair of opera glasses.

With the increasing popularity and use of the rotary high tension transformer the problem of current rectification (App. IV.) will vanish. The Coolidge tube alluded to in Chapter I. is, however, an effective means of avoiding the back E. M. F. of even the best induction coils. After twelve months' constant employment of these Coolidge tubes one would wish that they were more commonly in use, as their advent marks the greatest step made in radiography in recent years. The book is excellently illustrated, is an interesting volume for the expert radiologist, and is an essential and instructive treatise to every radiographer who wishes to exercise his art to the fullest degree, and a close perusal of the subject matter of the volume by the medical profession generally would throw light on what is, for some reason, the little understood, but immensely important, art of determining the position of foreign bodies in the human body.

NEW EDITIONS.

A Text Book of Pathology. By Alfred Stengel and Herbert Fox. Sixth Edition, Reset. Pp. 1024. Philadelphia and London: W. B. Saunders Co. 1915.

A text book which reaches its sixth edition in seventeen years is obviously one which fills a want. A book of this kind which is added to and altered from time to time as each edition is required is apt to lose some of its original continuity unless it be to a large extent rewritten. This has been done in the present instance, and the well-known Stengel's *Pathology* now becomes a *Text Book of Pathology* by Stengel and Fox. In its present form the book exceeds a thousand pages and possesses five hundred illustrations.

Text books of pathology, as of other sciences, may be divided into two categories. In the first place, those which are intended for students, which emphasise important points and explain difficulties, and which do not try to say something about every pathological condition; secondly, those which are encyclopædic, to which the specialist as well as the student turns when all available information on a subject is wanted, or when some rare thing is met with.

The present volume belongs decidedly to the second category. We are afraid that in reading the book the junior student would find it hard to differentiate between important and less important matter.

Some subjects receive rather scant attention. For example, acute liver atrophy is an important condition, not merely from its interest as a rare disease, but much more because of the chapters in general pathology which it illustrates, and the assistance which it gives to the understanding of liver cirrhosis. In the present volume the subject is dealt with in a few lines. The subacute forms of the disease are not mentioned at all, and the regenerative changes so constantly observed are barely referred to. Amœbic dysentery is dealt with in considerable detail, but no mention is made of the initial lesion—the flask-shaped ulcer with central slough—by which the disease may be differentiated from other forms of ulceration of the large bowel.

On the whole, however, the information which the volume contains is accurate, and, with the above limitations, the book can be recommended to students. The illustrations are for the most part very good. We recognise a number of old friends amongst them, some of which, by the way, are not acknowledged. Some of the coloured plates are particularly good.

BOOKS RECEIVED.

BAYLIS, WM. I. K. Therapy in Pulmonary Tuberculosis . . . (*J. Wright & Sons, Ltd.*) 3s. 6d.
BAST, E. D. The Dietetic Treatment of Diabetes. Seventh Edition
(*The Pencarrow Press, Allahabad*) Re. 1-8.
BELOU, PEDRO. Anatomia de los Conductos Biliares y de la Arteria Cistica
(*Buenos Aires*)
BISNELL, J. F. Manual of Operative Surgery. Seventh Edition (*H. K. Lewis & Co., Ltd.*) 32s.
BLACKSTONE, SIR J., and A. E. GILES. The Diseases of Women. Seventh Edition
(*Wm. Heinemann*) 15s.
THE Child Welfare Annual, 1916-7. Vol. I. Edited by T. N. Kelynack
(*Bale, Sons & Danielsson, Ltd.*) 7s. 6d.
CLAYTON-GREENE, W. H. Pye's Surgical Handicraft. Seventh Edition.
(*J. Wright & Sons, Ltd.*) 15s.
GIESLER, W. Laboratory Manual in General Microbiology . (*Wilcox & Sons, New York*) 10s. 6d.
GOULD, G. M., and R. J. E. SCOTT. The Practitioner's Medical Dictionary
(*H. K. Lewis & Co., Ltd.*) 17s.
GRAY, H., and R. HOWDEN. Anatomy: Descriptive and Applied. Nineteenth Edition
(*Longmans, Green & Co.*) 32s.
THE Johns Hopkins Hospital Reports, 1916. Vol. XVII. (*Johns Hopkins Press, Baltimore*)
MCDONAGH, J. E. R. Lectures on a Chain of Research on Syphilis (Oxidation and Reduction)
(*Hodges & Figgis*)
THE Medical Clinics of Chicago, 1916. Vol. I. No. . . . . (*W. B. Saunders Co.*)
NALL, S., and C. J. N. LONGRIDGE. Aids to Obstetrics. Eighth Edition
(*Bailliere, Tindall & Cox*) 2s.
OLIVER, G. Studies in Blood-Pressure. Third Edition . . (*H. K. Lewis & Co., Ltd.*) 7s. 6d.
REPORT upon the Work of Medical Inspection in the City of Carlisle for the Year 1916 . .
REPORT on the Sanitary Condition of the City of Carlisle for the Year 1916 . . . .
RIFFEL, J. R., and J. A. KELLY. Treatment of Fractures . . . (*J. P. Lippincott Co.*) 21s.
RUSSELL, HON. BERTRAND. Notes on the Causation of Cancer . (*Longmans, Green & Co.*) 3s. 6d.
SCHAFER, SIR E. A. The Essentials of Histology. Tenth Edition.
(*Longmans, Green & Co.*) 10s. 6d.
SIMPSON, W. J. The Maintenance of Health in the Tropics
(*Bale, Sons & Danielsson, Ltd.*) 2s. 6d.
SHAW, P. . . . The Diagnosis of Nervous Diseases. Fourth Edition
(*Edinburgh Medical*) 15s.
SHAW, P. . . . and A. EVANS. Nerve Injuries and their Treatment
(*H. Kimpton, Hodder & Stoughton*) 8s. 6d.
THE *Student*. Turner Memorial Number, 11th July 1916
(*Student Representative Council, Edinburgh University*)
TRANSACTIONS of the American Surgical Association. 1915. Vol. XXXIII.
(*W. J. Dornan*)
WALKER, W. . . . Post-Mortem Examinations . . . . (*W. B. Saunders Co.*) 21s.
YOUNG, . . . The Medical Profession Outside . . . (*H. K. Lewis & Co., Ltd.*) 3s. 6d.

EDINBURGH MEDICAL JOURNAL.

EDITORIAL NOTES.

A Hundred Years Ago

We have lately chanced on a *Report of Cases made in the British Military Hospitals at Brussels after the Battle of Waterloo*, by John Thomson, the first Professor of Military Surgery in Edinburgh, which is not without interest at the present moment.

"Upon hearing of the result of the battle of Waterloo," he begins, "I immediately resolved to proceed to Belgium, that I might have an opportunity of observing the medical and surgical condition of the men who had been wounded in that battle." Having obtained his credentials he left London on the 4th and arrived at Brussels on the 8th July, accompanied by Dr. Somerville, P.M.O. in Scotland. Every facility was afforded them for studying the wounded in the hospitals at Brussels, Antwerp, and Termonde. The official estimate of the wounded in the battle of the 16th and 18th of June was 8000, and "in consequence of the great number of wounded, some days were required for their removal from the field and their conveyance to Brussels." Those who could not be accommodated in Brussels were sent by canal to Antwerp. Several thousands of French wounded had been left on the field, and these were sent to Antwerp and Termonde.

We read much of the "symptomatic fever" which "put on much of the appearance of a bilious remittent or continued fever," and which "it deserves particularly to be remarked, began to prove fatal by the seventh day after the battle and continued to be so till the twenty-first day, when the number of deaths suddenly became fewer."

Hospital gangrene when once established in a ward seems to have spread with considerable rapidity, but curiously enough the writer expresses the definite opinion that he was "doubtful if it was ever communicated from one patient to another," and inclined to the belief that it was "endemical, and depended on the same causes as the fevers of the country."

No contagious fevers existed in any of the hospitals in Brussels or

Antwerp, which Thomson ascribes to the great attention paid to cleanliness and to ventilation in the British hospitals, and "to the habits and discipline of our troops with regard to personal cleanliness."

In view of the experience of the present war, it is interesting to read regarding tetanus, that "the number of those affected with that disease was inconsiderable, and in the greater proportion of instances in which it had occurred it appeared to be of a chronic or mild type . . . a form of tetanus in which recovery often takes place without much aid from medicine."

The following paragraph occurs in the section dealing with gunshot wounds: "No fear is now entertained, either by medical men or soldiers, of any kind of poison being introduced into the body by means of musket balls. All the consequences, immediate and remote, to which these balls give rise, are justly referred to the contusion, laceration, and division, occasioned by their impulse. Accordingly, surgeons now no longer think it necessary to dilate, cauterise, or suck such wounds for the purpose of destroying or extracting poison."

Secondary hæmorrhage "from the extremities of stumps after amputation, and from the canals of gunshot wounds," was of frequent occurrence, and usually took place from the twentieth to the thirty-fifth day. "In the dissection of the wounds and the stumps of those who had died of this hæmorrhage, I have not been able to detect, even with the aid of injections, the vessels from which the blood had been poured out in great profusion before death."

Then, as now, gunshot fractures of the femur weighed heavily on the minds of surgeons. Various attempts were made to apply continuous extension in such cases, but apparently without great success, for the writer sums up the discussion of the subject by saying:—"It must be left to future experience to determine whether, even in its simplest form, this apparatus is not a thing better calculated to amuse the rich, than to be of real service to the poor."

The following passage seems worth quoting as an historical parallel:—

"It would be unjust to the inhabitants of Brussels and Antwerp, to pass over in silence the humane and laudable attentions which they gave, both in public and in private, to our wounded men. From the commencement of the engagements, the deepest interest had been felt by the Belgians in the success of the British, and the heroism which had delivered them so suddenly, by a victory so decisive, from the terrors of invasion, excited in their minds the strongest emotions of admiration and sympathy. For some days after the battle, the inhabitants of Brussels, the city nearest to the scene of action, devoted themselves entirely to the care of the wounded, gave to them every assistance in their power, and were eager to receive them into their houses. On the day after the battle, as I have been informed by a

very intelligent eye-witness, the streets and squares presented a most singular and interesting spectacle. The shops were shut, the people were at their doors administering cordials, and offering dressing to the wounded, taking the tenderest care of them. The most delicate females, and people of all ranks, were occupied in this manner. Hundreds of wounded were to be seen in the streets, and some were to be found in every house. Even after the hospitals were fully established, several hundreds of privates, besides the officers, were voluntarily received and taken care of by the inhabitants during their cure. In the course of our visits to wounded officers, in private quarters, we had frequently occasion to observe the sacrifices which the inhabitants cheerfully made of their accommodations and comforts to their wounded guests, the personal services which they rendered, and the kindness they showed in presenting them with wine, fruit, and other luxuries. That this kindness proved injurious, in some instances, by interfering with the regimen and medicines prescribed by the medical officers, cannot be denied; and on this account it sometimes became necessary to discourage acts of benevolence which had the relief and comfort of the wounded solely for their object."

WAR HONOUR.

Victoria Cross.—The supplement to the *London Gazette* issued on 5th August announces that the King has conferred the Victoria Cross upon Captain JOHN LESLIE GREEN, R.A.M.C. (T.F.), for most conspicuous devotion to duty. Although himself wounded, he went to the assistance of an officer who had been wounded and was hung up on the enemy's wire entanglement, and succeeded in dragging him to a shell hole, where he dressed his wounds, notwithstanding that bombs and rifle grenades were thrown at him the whole time. Captain Green then endeavoured to bring the wounded officer into safe cover, and had nearly succeeded in doing so when he was himself killed.

CASUALTIES.

KILLED in action on July, Captain ALEXANDER PETRIE LOW, R.A.M.C. (T.F.).

Captain Low was educated at Edinburgh University, where he graduated as M.B. and C.M. with honours in 1896. After acting as resident medical officer in the Royal Infirmary, Edinburgh, he went into practice in Dundee, and was appointed surgeon of the Dundee Royal Infirmary. He was surgeon to the Seventh Highlanders at the front.

KILLED in action, Lieutenant RICHARD FENTON THEODORE NEWBERY, R.A.M.C., on 14th July, aged 25.

Lieutenant Newbery was educated at Edinburgh University, where he graduated as M.B. and Ch.B. in 1915, and, after acting as house-surgeon and house-physician of Edinburgh Royal Infirmary, took a temporary commission as lieutenant in the R.A.M.C. last October. He was attached to the Northamptonshire Regiment.

DIED of wounds, Captain WILLIAM GARROW SHAND, R.A.M.C., on 25th July, aged 34.

Lieutenant Shand was educated at Glasgow University, where he graduated as M.B. and Ch.B. in 1903.

DIED on service at sea, Dr. ELIOT WILLIAM WELCHMAN, on 15th July, while employed as a Surgeon in the Admiralty Transport Service.

Dr. Welchman took the diplomas of L.R.C.S. and L.R.C.P. at Edinburgh in 1883, and the M.R.C.S. in the same year, after studying at Queen's College, Birmingham.

KILLED in action, Captain JULIUS HENRY BEILBY, R.A.M.C. (T.F.), on 23rd April.

Captain Beilby was educated in the medical schools of the University and Royal College of Surgeons, Edinburgh, graduating as M.B. and C.M. in 1889, and afterwards studying at Vienna. He was medical officer of the Worcestershire Yeomanry (Queen's Own Worcestershire Hussars).

KILLED in action, Temporary Lieutenant BENJAMIN RICHARD ROBERTS, R.A.M.C., reported in the casualty list published on 8th August.

Lieutenant Roberts was educated at Queen's College, Galway, and at the Royal College of Surgeons, Edinburgh, taking the Scottish Triple Qualification in 1898. He was in practice at Johannesburg when the war broke out.

MEDICAL STUDENTS.

KILLED in action, Second-Lieutenant W. J. DUNN, King's Own Scottish Borderers.

Lieutenant Dunn was studying medicine at Edinburgh University before the war, and had been at the front for six months.

KILLED in action, Second-Lieutenant ROBERT KENNEDY, Royal Scots Fusiliers, aged 20.

Lieutenant Kennedy had just completed the first year of his medical course when he received his commission in June 1915.

DIED of wounds, Second-Lieutenant J. LESLIE LAIDLAW, Argyll and Sutherland Highlanders.

Lieutenant Laidlaw had completed the second year of his medical course at the University of Edinburgh.

DIED of wounds received on 1st July, Captain DESMOND MCLLALY, Northumberland Fusiliers.

Captain Mullaly before the war was a medical student at Glasgow University, where he was in the O.T.C.

KILLED in action on 28th June, Corporal JAMES FLEMING STEWART, Royal Engineers, aged 22.

Corporal Stewart was a medical student at the University of Edinburgh.

KILLED in action, Lieutenant LESLIE R. S. GUNSON, Royal Garrison Artillery, aged 21.

Previous to the war Lieutenant Gunson was a medical student at Edinburgh University.

KILLED in action in July, Second-Lieutenant JOHN W. M'VICKER, King's Liverpool Regiment.

Lieutenant M'Vicker had completed the first year's medical study at Edinburgh University on the outbreak of the war.

TRIPLE QUALIFICATION.

At the recent examinations of the Board of the Royal College of Physicians of Edinburgh, Royal College of Surgeons of Edinburgh, and Royal Faculty of Physicians and Surgeons of Glasgow, the following candidates passed the *First Examination*.—Edward J. Allan, Andrew S. Irvine, Andrew W. Smith, and Peter E. Malloch.

Second Examination.—Thomas Blaney, Gordon S. Woodhead, Hugh W. Howatson, Mayberry H. Carleton, Thomas F. Mumford, Harold C. Smith, and Buenaventura Ramirez.

Third Examination.—Bernard M. Lynam, Don A. Walpola, Daniel C. Howard, William B. Watson, Mahmoud A. K. Mofreh, Donald Stewart, and William E. Mason.

Final Examination.—The following candidates having passed the Final Examination were admitted L.R.C.P.E., L.R.C.S.E., L.R.F.P.S.G.—Thomas Downie Rennick, Shapoor Dashaw Varma, Frank Bertram Macaskie, Maurice Andrew White, Arthur Leslie Gubbin, Phyllis Hester Brooke Coghill, John Ross, John Edward Kitchen, and Ethel Mary Dukes.

THE HUNCHBACK OR GIBBUS PELVIS (KYPHOTIC PELVIS; KYPHOSIS PELVIS).

By D. BERRY HART, M.D., F.R.C.P.E.,
Lecturer on Midwifery and Diseases of Women, Surgeons' Hall, Edinburgh.

Preliminaries as to Nomenclature. — The usual term, that of kyphotic pelvis, is a complete misnomer, as it is the spinal column and not the pelvis that is kyphotic or hunchbacked. This pelvis has its poise and diameters altered; the sacrum is displaced up and back with the coccygeal end swung forward, but all this is due to the spinal kyphosis and is not part of it unless in the lumbo-sacral form. The terms I propose, that of hunchback or gibbus pelvis, means the pelvis caused by hunchback or gibbus. Both of these terms, hunchback and gibbus, are used as nouns and express a causal relation. Gibbous is an adjective, and erroneous, like kyphotic. The term kyphosis pelvis, proposed and used by Breus and Kolisko, is correct, and an equivalent to those I have employed in the title.

History of the Recognition of the Changes in the Hunchback Pelvis. —The first to point out the elongation of the brim-conjugate was Herbiniaux of Brussels in 1794. In his *Traité sur divers accouchemens laborieux, etc.* (vol. i. p. 270), he remarks: "302. Cependant nous devons que ces vices de conformation ne rendent pas toujours impossible l'accouchement naturel et qu'an contraire ils le favosient quelque fois, comme, par exemple, quand la gibbosité est vers les vertebres lombaires. N'est facile de concevoir que les vertebres des lombes étant alors portées en arriere, la partie supérieure de l' os sacrum a dû les suivres, de forte qu'il en résulte un aggrandissement de la cavité du petit bassin vers le haut; c'est le cas où l' os sacrum est ordinairement redressé" (*op. cit.*, vol. i. p. 270, par 302).

Rokitansky in his "Pathological Anatomy" (*Syd. Soc. Trans.*, 1850) pointed out the relations between spinal kyphosis and the pelvis (*op. cit.*, vol. iii. p. 241), drawing attention to the lower compensatory spinal lordosis. He considered that the adult pelvic deformity is the primary condition and the spinal one consecutive (vol. iii. p. 241), an error, as we shall see, but states correctly that "the pelvis in angular curvature is generally very capacious; its height is considerable, and the pre-

dominant diameter is manifestly the conjugate. The conformation of the pelvis chiefly arises from the diminution in size of the abdominal cavity, which is produced by the depression of the thorax" (*op. cit.*, p. 243).

Freund, sen., held that the pelvis was gibbous in the fœtus, and that the adult kyphosis was secondary to it, an interesting but erroneous opinion.

Excellent papers have been contributed by Klien, Freund, sen. Neugebauer, sen. and jun., and also by Chantreuil and Tremer, but the most outstanding and penetrating investigation was published in 1865 by Breisky, whose work will be considered later.

In our own country Champneys (1883) and Barbour (1894) have been the principal investigators. Barbour's *Atlas* is a great and unique contribution to the facts, as he investigated not only the pelvis but also the sagittal mesial and lateral sections of the unopened thorax and abdomen of a cadaver with marked kyphosis, and made a most notable addition to our knowledge.

Champneys investigated the question of this deformed pelvis in its obstetric aspect, and also, in another paper, made more clear the nature of femoral pressure.

Finally, on this head we may note that Breus and Kolisko gave a most comprehensive account of this deformity in their monograph on "Deformed Pelves" (*Die pathologischen Beckenformen*, Deuticke, 1904).

Anatomy of the Hunchback Pelvis.—In studying this deformity I have had the advantage of examining two specimens of my own and the examples of it in the Museums of the Edinburgh University and of the Royal College of Surgeons of Edinburgh, and also the plates and descriptions in the various monographs, text-books, and papers given in the literature.

The Kyphotic Spine.—Hunchback.—It must be briefly noted that spinal kyphosis means a backward angular projection of the spine at various levels. The projection of the spine is evidenced by a dorsal convexity, and is the result of tubercular caries leading to destruction, more or less evident, of the bodies and processes of the vertebræ, the laminæ excepted. The spinal cord is not, as a rule, compressed. The kyphosis may be pure or associated with compensatory lordosis when at higher levels, or complicated with scoliosis leading to mixed deformities—kypho-scoliosis, for instance. These have been specially considered by Leopold and others, and their

consideration need not detain us at present. An important generalisation is that the higher the kyphosis the less the effect in distorting the pelvis. Thus lumbo-sacral kyphosis causes the most marked form of hunchback pelvis; next comes the lumbar and lumbo-dorsal forms. When the kyphosis is high up in the dorsal region, or when in the dorso-cervical region, a compensatory lordosis below may so counteract the upper kyphosis that the pelvis may remain fairly normal. Barbour's plates may be studied in this connection.

Klien states the proportion of labours in those with kyphosis pelves to normal ones as one in six thousand and sixteen (1 : 6016); that lumbo-sacral kyphosis is as frequent as lumbar and lumbo-dorsal together, and that pelvis obtecta (Fehling), *i.e.* the lower limb of the kyphosis projecting over the brim, exists in about 8 per cent. Klien's statistics show further that three-quarters of all such labour cases end at term and one quarter prematurely. About one-half of the labours were spontaneous.

The prognosis as to full-time labour depends on the transverse ischial tuberosity measurements, and about 8 cm. is the lower limit for ordinary delivery. This diameter is not the only criterion, as the available outlet space is really the triangular one mapped out by three points, viz. the ischia and the tip of the coccyx (Champneys) (Fig. 2).

Late kyphosis, after the pelvis is ossified, may leave the pelvis unaffected (see Breus and Kolisko, p. 168, Fig. 41).

The special characteristics of the hunchback pelvis may be classified as follows (Fig. 3):—

(1) The high position of the promontory and the flattening of the angle of the conjugate to the horizon; the changes in the sacrum.

(2) The changes in the brim diameters and upper pelvic strait.

(3) The changes in the lower strait and outlet.

(1) *The High Position of the Promontory and the Flattening of the Angle of the Conjugate to the Horizon; the Changes in the Sacrum.*—In almost all hunchback pelves the promontory *simulates high lumbo-sacral assimilation*, as the first sacral vertebra is on a higher level relative to the top of the iliac crests than in the normal pelvis, but the number of sacral vertebræ is still five. There is thus no *high assimilation* of the promontory, but a *higher position*, due, as we shall see, to purely mechanical causes.

The *pelvic brim*, or rather the angle the brim conjugate makes with the horizon, is lessened, usually considerably in the cases of

lumbo-sacral, lumbar and lumbo-dorsal curvatures, but in the high dorsal and cervical may be normal. The *sacrum* is elongated, narrowed, and has a lessened curvature. The site of the original intervertebral disks is greater vertically, and this ossific union is rarefied, with many spicules attached above and below to the vertebral edges. The first sacral vertebra is bent back somewhat, and thus the anterior sacral curvature is *f*-shaped.

(2) *The Changes in the Brim Diameters and Upper Pelvic Strait.*—The *brim diameters* are increased and the upper strait much more capacious than in the normal pelvis, as will be seen in

Figures 1, 2, and 3. Thus there is no obstruction to labour in the upper strait, and the head at the beginning of labour lies deep down in the cavity, usually in a transverse position.

(3) *The Changes in the Lower Strait and Outlet.*—It is here that the obstruction, if any, to labour exists. The *side walls* of the pelvis converge, the *pubic arch* is narrow, the *ischial tuberosities* nearer, and thus the *pelvic outlet* becomes often greatly diminished. This diminution is increased by a *transverse rotation of the sacrum* such that the tip of the coccyx is tilted forward.

Some *minor points* may be noted here, viz. the more upward direction of the sacral formina; the increased size of the anterior inferior spinous processes; the flattening of the ilia; and the throwing out of the anterior superior spinous processes. The

vulva in the erect posture looks more forward than in the normal woman, and the whole figure is squat, as is well shown in one of Freund, sen.'s, clinical cases (*Atlas*, Plates 19 and 22).

Explanation of the Changes in the Hunchback Pelvis.

The changes to be explained are—

(*a*) The flattening of the brim-conjugate angle; the high position of the promontory.

(*b*) The increase in capacity of the upper pelvic strait.

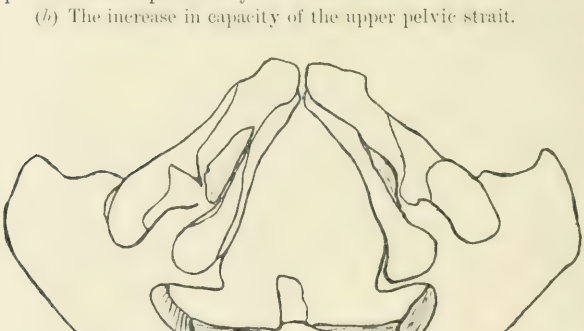


Fig. 2.—Tracing of Outlet of Hunchback Pelvis.

(*c*) The changes in the sacrum.

(*d*) The convergence of the pelvic side walls and the diminished outlet diameters; the narrow pubic arch.

(a) *The Flattening of the Brim-Conjugate Angle; the High Position of the Promontory.*—In the normal upright posture of the body we have the weight of the trunk, arms, and head resting on the sacrum. The sacrum is not a wedge in function, as its anterior transverse diameter is wider than the posterior. Matthews Duncan considered this carefully, and showed that at the sacro-iliac synchondrosis there was a bite or "joggle" between the sacrum and ilium, and that the trunk and this tie-beam were suspended by the strong superior sacro-iliac ligaments. This weight, acting down, is resisted by the legs acting in a line joining the foot and acetabulum.

The pelvis has an inclination normally to the horizon of from 60° to 100°, and we may regard this as follows: —

The pelvic inclination begins after the child learns to walk. The lumbar curve is formed, and then the balance of the child and adult is such that a perpendicular from the centre of gravity of the body drops a little behind the transverse acetabular diameter or on the top of the heads of the femora. The normal tilt of the pelvis is due to the lumbar curve, and is so balanced that any tendency of the trunk to topple back is resisted by a check element on the part of the anterior ilio-femoral or Y-shaped ligaments of Bigelow. This balance varies according to certain conditions, and may be increased or lessened abnormally when the pelvis is rickety; the lordosis of the lumbar curve is much increased, and we get the pelvic inclination approaching the vertical, and telling more directly on the conjugate so as to narrow it. On the other hand, as we have seen, in the hunchback pelvis, where the brim-conjugate is elongated, the brim-conjugate angle is usually much diminished.

The normal pelvis can have its brim-conjugate increased by extreme straddling of the legs and turning out of the toes to the utmost. The result of this manœuvre is thus to tilt the pelvic brim towards the vertical, and increase the tension on the ilio-femoral ligaments, which, fixed below at the anterior inter-trochanteric line and above at the anterior inferior spinous processes, naturally tilt the pelvis upward and forward, the pelvis rotating on the heads of the femora.

On the other hand, the turning in of the toes with slight divergence of the limbs diminishes the pelvic brim inclination, owing to the diminished tension on the Y-shaped ligaments.

In the hunchback pelvis the centre of gravity of the body is further back in the sacrum, or may, indeed, be in the lower limb of the kyphosis. Thus the vertical from it has greater leverage, and consequently the brim-conjugate angle becomes diminished.

The high position of the promontory is present in the great majority, but not in all of the hunchback pelves (see p. 152).

The question of assimilation of the promontory, normal and abnormal, must therefore be considered, as it is of importance in regard to deformed pelves, and has hitherto been little considered in this country.

By assimilation of the promontory we mean how the promon-

tory is formed — whether by the first sacral and last lumbar (normal symmetrical assimilation) or by the third and fourth lumbar (high symmetrical assimilation), leaving out of the question at present unilateral assimilation. Normally, the sacral vertebræ are welded together, and the first sacral with the non-welded 4th lumbar forms the promontory. In high assimilation the 4th lumbar is welded with the sacral vertebræ and the promontory formed by the 3rd and 4th lumbar vertebræ. When the latter occurs without complication we get what is known as the funnel-shaped pelvis, where we have an increase in the brim capacity but narrowing in the transverse of the outlet, with some convergence of the pelvic side walls.

Probably what lies behind this question is that of the formation of the sacro-iliac joint. Of this we know little, but if the sacro-iliac joint forms in the region of the two upper sacral vertebræ we get normal assimilation, but if between the last lumbar and first sacral, high assimilation is the result. This question, however, requires investigation, as well as that of the varieties of welding above mentioned.

Sometimes we get low assimilation, four sacral vertebræ forming the sacrum. One of my specimens has high position of the promontory and four sacral vertebræ.

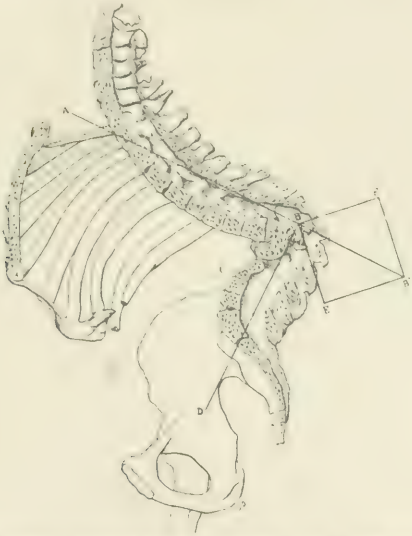
From the point of view of variation it is to be noted that in the vast number of cases we get a preponderance of normal assimilations, a certain much smaller number of high assimilations, and a much smaller number of low assimilations, this being analogous to an elementary frequency polygon, as we find in the summing up of all variations.

The high position of the sacral promontory in the hunchback pelvis is not a true high assimilation, but a higher position, as the number of the sacral vertebræ is not increased. There is not, therefore, congenital high assimilation in such, but a simulation of it.

(b) *The Increase in Capacity of the Upper Pelvic Strait.*—It is a well-known fact that the upper strait of the hunchback pelvis is in pure cases much enlarged. This is due to the lengthened conjugate and transverse and the higher position of the promontory, than in normal cases, as well as to the bending back of the 1st sacral vertebra. Two views may be taken of the significance of the high position in producing this enlargement, viz. that it is due to a passage up of the sacrum owing to the disturbance of the body poise caused by the kyphosis, or that this high position is

a high assimilation, and is really a primary condition of the pelvis. The former seems to me the correct view.

Fig. 3, from Tarnier, Budin and Chantreuil, expresses the first view, Chantreuil being the collaborator who wrote specially on kyphosis. The upper and lower dotted lines are drawn in the bony axis of the limbs of the kyphosis (A B, B C), and on A B produced the parallelogram of forces is erected. B D is resolved



into B F upwards and backwards and B E downwards and backwards. The two resolved forces thus give upward, downward, and backward components.

The serious objection to this explanation is that in the transmission of weight the *shape* of the supposed line of transmission is not to be considered. The line of transmission is that joining the pull and the resistance point. If we put ? on the centre of gravity for a kyphotic case, then the dropped vertical from ? will rotate the pelvis down, back, and up. An analogous case is that of axis-traction forceps. The line of pull is the straight line join-

ing the fist at the traction handle and the upper application of the traction rod. It would be erroneous to hold that the traction rod long axis above the bend of the rod expressed the downward and forward motion of the head, and the long axis of the traction rod below the bend the downward and backward action of a traction during a pain. What really happens in the hunchback pelvis seems to be this. Owing to the kyphosis the centre of gravity of the body is displaced backwards behind the normal centre. Thus the hunchback pelvis has its pelvic brim angle flattened. The pelvis is under three tensions, a downward, backward and upward, and a resisting one, due to the resistance of the ilio-femoral ligaments. Thus the conjugate is elongated and flattened. This can be illustrated as follows:—If one takes an ordinary ring pessary, placing an index finger at each end of a diameter, traction in opposite directions will elongate this diameter and make the ring an oval—that is, elongate the diameter under traction. There is also an upward displacement of the promontory.

If we erect the parallelogram of forces, as in Fig. 3, not on the limbs of the kyphosis, but on the brim-conjugate, so that it has the same inclination as in Fig. 3, there will be a downward and backward resultant, B B', the components being B F up and back and B E down and back. When the ilio-femoral and sacro-sciatic ligaments are at check, the up and back will still act at the sacro-iliac joints, the down and back being hindered.

The upper strait enlargement of the hunchback pelvis is thus due (1) to the elongation of the brim-conjugate as already explained (p. 156); (2) to the high position of the promontory, giving a conjugate, not from the symphysis pubis to the promontory in its usual relation to the top of the iliac crests, but to the higher promontory; (3) to the increase in the transverse of the brim as the higher position of the sacrum forces the upper ends of the ilia a little out (see p. 156).

(c) *The Changes in the Sacrum.*—These are (1) elongation, narrowing and straightening of the sacrum; and (2) a rotation of the sacrum on a sacral transverse axis, so that the tip of the coccyx goes forwards. Of course the elongation of the brim-conjugate retroposes the sacrum.

(1) The elongation, narrowing, and straightening of the sacrum are probably due to the tension on the sacrum from the downward, backward, and upward action of the kyphosis, resisted by the tension of the sacro-sciatic ligaments.

(2) The retroposition of the sacrum is not only a retro-translation as a whole, but also a rotation owing to the resistance of the sacro sciatic ligaments, and therefore the rotation as this resistance acts.

(4) *The Convergence of the Pelvic Side Walls and the Diminished Outlet; the Narrow Pubic Arch.*—The convergence of the side walls is due to the higher position of the promontory. The transverse measurement between the upper ends of the iliac bones is less than that between the normal sacro-iliac synchondroses, and thus the upper iliac bone ends are somewhat forced out by the high promontory. Owing to the symphysis joint resistance to this and the articulation of the innominate bones with the femora, the result is a rotation of the ischial tuberosities in, the convergence of the side walls from above down necessarily following. The narrow pubic arch follows from the above explanation.

The explanation of the changes in the hunchback pelvis due to the lower kyphosis is evidently a mechanical one, as already narrated, and was, in the main, clearly stated by Breisky in 1885.

I consider that the high position of the promontory is part of the resolution of the forces brought into play by the disturbance of the pelvic poise due to the kyphosis. Undoubtedly the synchondrosis joints are slackened, the surfaces eburnated, and the characteristic ear-shaped appearance straightened out, so that the upper and lower portions of these surfaces meet at a much smaller angle.

We have, therefore, evidence in these local changes that these points are the bearing points where such alterations take place, and the eversion of the anterior symphysis edges found in the hunchback pelvis is evidence of an anterior bearing-point. We may divide the bearing places for the pelvis in kyphotic spine into two: (1) The hip-joints involved in the flattening of the brim inclination; and (2) the sacro-iliac joints, the locus for the sacral retroposition and high position.

Summary.—1. The so-called kyphotic pelvis is more accurately termed the hunchback or gibbus pelvis (not gibbous).

2. The cause of the hunchback pelvis is kyphosis of the spine in the lower spinal or spinal and sacral regions.

3. All the changes in the poise of the hunchback and in its various pelvic strata can be explained on the mechanical principles already given, and these were first, in the main, clearly given forth by Breisky.

There is nothing developmental in the pelvic changes found in the hunchback pelvis.

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CLINICAL RECORDS.

REPORTS AND COMMENTARIES ON FIVE SURGICAL CASES.*

By J. CRAWFORD BURNS, Student of Medicine.

Case I.—Paget's Disease of the Nipple and Carcinoma of the Breast, with Special Reference to the Clinical Features and Pathology of the Condition.

Mrs. M. B., aged 42 years. Admitted to Ward 9, Royal Infirmary, on 13th October 1915, complaint being a "sore on the breast."

History.—She stated that the condition began in 1912, and was of gradual onset. The first thing she noticed was a scab on the point of the nipple of the left breast. This scab took some months to develop and separate; the nipple was somewhat red, and different in colour from the other. She said it was not sore, and there was no feeling of heat or throbbing in the affected part. The scabbing appeared and disappeared at short intervals, and throughout the whole time the area of areola affected became larger and larger. The patient never experienced any systemic disturbance, and she said she had not lost weight. Then three weeks before admission, 23rd September 1915, the patient noticed a lump in the breast which was somewhat painful on pressure. On her doctor's advice she came to the Royal Infirmary for further examination and treatment.

Examination on Admission.—Inspection.—The areola of the left breast was enlarged, and round the periphery was a narrow zone of dry, yellow, scaly material. In the centre of the areola there was a more florid granulated area and the nipple was almost indistinguishable. On close examination the surface epithelium of the areola appeared to be excoriated, and little scaly patches

* Being "the report and commentary on cases treated in the general surgical wards of the University professors and teachers in the Royal Infirmary," for which the Pattison Prize in Clinical Surgery was awarded in 1916.

could be made out. The periphery of the affected area was distinct and fairly uniform, the whole patch being somewhat raised above the general skin surface.

On grasping the nipple and areola a distinct feeling of thickening could be made out. There was no noticeable discharge, and the patient did not complain of itching.

Palpation.—On palpating the breast a hard mass was found in the lateral half, which was movable over the pectoral muscles below, but adhering to the skin. It was of stony hardness, and a little tender on pressure. The glands in the axilla were not definitely palpable, and no enlarged supraclavicular glands could be made out.

Her *family history* showed nothing of interest. Her menstrual periods were still regular, and she had had no abscesses in the breast.

Treatment.—Operation.—It was decided, in view of the evident malignant condition of the breast, to operate and excise the breast and affected lymph glands.

Operation performed by Mr. Miles, 15th October. The anæsthetic used was chloroform, followed by ether.

The left arm was fully abducted by an assistant, and an incision was made, beginning at the clavicle and passing over the pectoral muscles to the posterior fold of the axilla. By dissection, chiefly gauze dissection, the axilla was cleared, and the glands along the scapular vessels were also removed.

An elliptical incision was made surrounding the breast. The breast with the tumour was removed, along with the superficial part of pectoralis major. The breast and axillary fascia and glands were removed together. A drainage-tube was passed through the axillary fold and the edges of the wound approximated and sutured by horsehair. The skin round the margins of the wound was scarified and dressing applied. The breast was bandaged with the arm in the abducted position, with plenty of cotton-wool in the axilla. The arm in bed was supported with a pillow.

Progress Notes.—Patient soon recovered from the effects of the operation and anæsthetic, and had slight vomiting. She complained of a certain degree of pain, which was relieved by a hypodermic injection of heroin, $\frac{1}{12}$th grain.

17*th October.*—Wound was dressed.

8*th November.*—Patient went home. During this time there was no rise in temperature, and recovery was uninterrupted.

Fig. 1.—Photograph of Breast showing the Condition of the Nipple

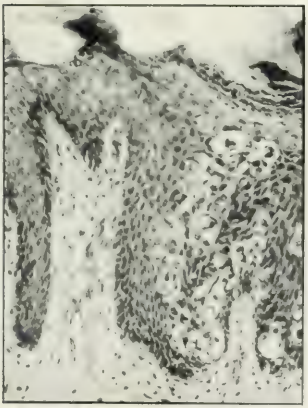


Fig. 2.—High Power View of Section of Areola

The breast excised at the operation was preserved, and sections were made of the gland and of the nipple.

Commentary.—The diagnosis was that of Paget's disease of the nipple with carcinoma of the breast, and this was verified by the microscopic examination of the tissues.

The history and clinical appearances are typical of such a condition. It is found in women between the ages of 40 and 60. The clinical features correspond closely to the description given by Sir James Paget in 1874, in which he says that "the patch had the appearance of a florid red raw surface, very finely granular, as if nearly the whole thickness of the epidermis were removed, like the surface of a very acute, diffuse eczema, or like that of an acute balanitis." Paget also pointed out that this condition of the mammary areola was followed by the appearance of cancer in the affected breast.

Paget's disease of the breast is, however, comparatively rare, for carcinoma of the breast is only preceded by this condition of the nipple in about 1 per cent. of cases. In the early stages the appearance of the nipple gives rise to the probability of it being an acute eczema. The points of difference, however, are:—(1) Absence of vesicles; (2) absence of excessive itching; (3) uninfluenced by ordinary remedies.

Two forms of Paget's disease of the nipple may be met with clinically:—(*a*) A wet form; (*b*) a dry form.

In the former there is a discharge, viscid but not purulent, the drying of which gives rise to scabs.

In the dry form there is no such discharge, and when the areola is pressed between the finger and thumb it gives a crackling sensation. As in this particular case the nipple may be indistinguishable, owing to ulceration.

The time that elapses between the appearance of this condition of the nipple and areola and the appearance of cancer in the breast varies, being anything from 2 to 10 years. The tumour in the breast is clinically a scirrhus cancer.

Pathology.—This aspect of the case is also one of great interest. Following on the work of Paget a good deal of work was done on the morbid histology of the condition, and much controversy seems to have arisen among the earlier investigators as regards the true pathology. Some came to the conclusion that the disease was a "malignant dermatitis" produced by the presence of protozoa in the epidermis, these protozoa being in the nature of psorosperms or coccidiæ. Later workers, however, showed that

this was not the case, and that the psorosperms were but the result of degenerative changes in the chromatin of the malignant epithelial cells.

Examination of the Section from Areola.—There is seen, first of all, on the skin surface epithelial débris, bacteria, and masses of inflammatory cells. Passing into the deeper layers of the skin we see the projecting papillæ covered by the rete Malpighi. The germinal layer of cells is distinct, and we can trace their transition into prickle cells. Then we have a somewhat abrupt change to clumps of large clear cancer cells (Paget cells). This is best seen in the interpapillary area of the rete Malpighi. These cancer cells by their proliferation have caused pressure on the surrounding prickle cells, causing them to appear crushed, and in some cases atrophied. In some areas the cancer cells have passed down among the prickle cells to the germinal layer.

Under the high power these cancer cells are easily picked out. They are large, and for the most part oval cells. Very noticeable are the prominent nucleus and chromatin threads. The body of the cell appears clear, due to the very faintly staining protoplasm. The cell membrane, however, is more distinct. Mitotic division is seen occurring in various cancer cells. These proliferating cells, which are atypical epithelial cells of a malignant character, are inserted among the normal prickle cells.

More superficially in the section we see an infiltration of the corium by small cells, and in one or two places are small abscesses. In the fibrous stroma of the papillæ there is a certain degree of perilymphatic cellular infiltration. In the case of the nipple, malignant epithelial cells appear in the peridueal tissue of the mammillary duct.

Examination of Section from Breast.—In this section the most noticeable thing is the blocking of the ducts with masses of proliferating cancerous epithelial cells. Throughout the section this holds good, and the lymphatics are also seen to be invaded with these cells. The acini are not affected to any extent, and the main change is to be found in the ducts. The cancer cells appear to be growing also beneath the lining epithelium of the duct, and there is some spread to peridueal tissue. We have here, evidently, a duct carcinoma.

Summary of the Case.—We have, therefore, a carcinoma of the skin of the nipple and areola, this giving rise to a chronic inflammatory process simulating a chronic eczema, with inflammatory infiltration of the sub-epithelial layers. As time goes on, the cancerous

process reaches the lactiferous ducts, and the proliferating cells pass downwards along the ducts into the gland tissue of the breast, giving rise ultimately to a malignant growth of the nature of a duct cancer.

This information, derived from a careful study, both clinical and pathological, shows the case to be one of Paget's disease of the nipple with carcinoma of the breast.

Why cancer should develop in the breast in cases of Paget's disease of the nipple is not easy to explain, until we consider the strain upon such an organ as the mammary gland in the various periods of life—puberty, gestation, lactation, and senile changes —and this is particularly true of parous women.

Those changes, although physiological processes, are not far removed from the pathological. In accordance with its functions there are rapid proliferative changes in the breast tissue, especially the epithelial element, and any extra strain or source of irritation might well upset the balance between the physiological and the pathological.

In Paget's disease of the nipple there is a source of chronic irritation which might well account for the onset of a pathlogical process.

Case II.—Dermoid Cyst of the Orbit.

Margaret S., schoolgirl, aged 12 years. Complaint, "seeing double." Admitted to Ward 9, November 1915.

History.—About six years ago patient struck her head against a lamp-post, and from that date a lump appeared over the upper and anterior part of the right temporal fossa. This seemed to disappear at times and then to reappear. It was occasionally painful locally, and there was some pain just above the level of the lobule of the right ear and over the right cheek. There was never any pain over the supra-orbital nerve or over the infra-orbital foramen, and never any neuralgia or toothache. There was never any vomiting or headache. The swelling did not appear to have been increasing in size as far as the superficial part was concerned, but during the two weeks prior to admission there was a gradual development of diplopia. There was no motor or sensory disturbances in relation to any of the cranial nerves outside the orbit as far as could be ascertained. The patient did not notice any loss of vigour, and she did not think she had lost weight. Her appetite and powers of sleep were not disturbed.

There is no history of similar swellings in other parts of the body.

Pulse, 88; respirations, 24; temperature, 98·2° F.; heart and lungs normal.

Previous Illnesses.—Scarlet fever, measles.

Family History.—Father alive and well. Mother also alive and in good health. Three brothers and one sister, all in good health.

Examination.—There was a marked fulness over the upper part of the right temple, and there was nothing similar on the left side. There was exophthalmos of the right eye, together with a degree of internal and downward rotation of the eyeball. The pupils were equal, of normal size, and reacted to light and accommodation. The lateral movements and those in an up and down direction were somewhat limited, but the disability seems to have been due to a mechanical rather than a nervous cause. Von Graefe sign positive. The eye was protruded forwards and outwards. The fundus showed nothing except a slight degree of fulness of the veins, suggesting difficulty of return of blood. The vision was nearly full in that eye. The swelling in the region of the orbit was not definitely tender, and did not show signs of inflammation. The upper part of the tumour was of bony hardness, and was continuous with the anterior surface of the frontal bone. Below, the bony surface ended abruptly in a sharp edge well above the zygoma. Below the edge, again, the lower part of the tumour appeared to be soft and elastic. The whole tumour was above and distinct from the zygoma.

X-ray Appearances.—A tumour was seen involving an area of the orbit, cranial fossa, and temporal fossa on the right side of the skull. The edge of the tumour was seen to be dense and the centre quite clear. These points can be made out by reference to the print taken from the radiogram (Fig. 3).

Treatment.—1. *Medicinal.*—Potassium iodide in three-grain doses, administered three times a day, was tried, but no definite signs of improvement manifested themselves.

2. *Surgical. Operation.*—Anæsthetic used was chloroform, followed by ether.

A curved incision was made about 2 inches long over the temporal and frontal areas; a flap of integument was turned down. The tumour was easily made out, as the bone over it was as thin as parchment. It was opened and found to contain yellow fluid, with thicker yellow-like material in it. The tumour was found

Mayf. Sibbald
Dermoid Cyst of the
Orbit.

[illegible]

to be pushing forward the eye, which resumed its normal position when once the fluid was evacuated and the tension relieved. Posteriorly was dura mater. The cyst was emptied and the thin bone over it removed. The pericranium and temporal muscle were well drawn over it, and a small rubber-dam drain left in the superficial tissue. The wound was closed, dressings were applied, and the girl taken back to bed.

Progress Notes.—10*th December* 1915.—Stitches out; wound clean and dry.

13*th December* 1915.—Patient got up; progress satisfactory.

25*th December* 1915.—Patient went home.

Commentary.— In this case we had to deal with an orbital tumour of the nature of a dermoid cyst. In regard to its clinical features the girl gives a history of trauma—a blow on the head six years ago. It was following on this accident that the swelling was first noticed. How much the injury had to do with the appearance of the tumour is difficult to say. A certain amount of swelling may have occurred a day or two later as a result of contusion. Again, more particular attention may have been brought to bear upon this region, by the fact that the patient had received an injury, and a local condition which may have escaped earlier notice now became manifest on closer examination. It will be noticed there were no cerebral complications, as evinced by the absence of vomiting, headache, giddiness, etc. The growth of the tumour was very gradual, and was unattended by ill effects on the patient. There was no loss of weight; appetite was good, and sleep had not been interfered with. There was no history of involvement of any cranial nerves. About two weeks before admission a gradual increasing degree of exophthalmos was noticed on the affected side, and the patient complained of diplopia. The examination of the eye showed that the protrusion was due to mechanical means, a mass in the posterior wall of the orbit causing increased intra-orbital pressure. In this case the possibilities that had to be considered from the point of view of diagnosis were (1) gummatous periostitis; (2) traumatic cephalo-hydrocele; (3) sarcoma of the orbit; (4) dermoid cyst.

As regards the probability of gumma there was no improvement on a course of potassium iodide.

In the case of traumatic cephalo-hydrocele the accident that this girl sustained may have conceivably caused a fracture of the bone and injury to the dura mater, with the resultant escape of some cerebro-spinal fluid into the scalp, and this swelling kept up

by effusion of fluid as occurs in cases of traumatic cerebral œdema. Although such a condition is possible, it is not probable in this case. The accident does not appear to have been sufficiently severe to produce the necessary lesion, and there was no pulsation in the tumour as would have been met with in a spurious meningocele.

Sarcoma.—This appeared more probable. Three origins of sarcoma are available in this area—periosteum, diploe, and dura mater. In a periosteal sarcoma, a tumour of considerable size would have appeared fairly quickly, and would have been of a soft and pulsatile nature; also, there would have been evidence of general effects on the system—loss of weight, and involvement of the optic nerve and eyeball. If it was a sarcoma arising from the diploe, it would be of the nature of a myeloid sarcoma. In this case growth need not be rapid. Such a type of tumour is covered by a thin shell of bone, giving an egg-shell-like crackle on pressure, and the skin would become involved in the process of growth.

Sarcoma arising from the dura mater and penetrating the bone is more easily discounted, for well-marked cerebral symptoms appear even before a tumour mass is seen externally.

The diagnosis, therefore, appears to be a dermoid cyst. (*a*) It was a tumour of slow growth; (*b*) absence of constitutional disturbance; (*c*) position—a common site for dermoid is the outer angle of the orbit, which corresponds to an embryonic cleft; (*d*) rounded swelling, definitely limited, and the skin freely movable over it.

The history says that the swelling was first noticed six years ago. This type of tumour is congenital, but the tumour mass is not necessarily seen from birth. In this case the girl was 6 years of age, and her tissues were reaching a period of increasing potential activity, and the sequestrated portion of epiblast capable of becoming rapidly proliferative.

Position of the Tumour.—This explains the origin of the dermoid in this region. Here at the outer angle of the orbit there occurs fusion of the embryonic orbital-nasal cleft. When the embryonic segments fuse the epithelium should normally disappear, but in a certain number of cases this disappearance of surface epithelium is incomplete and a portion is sequestrated, the growth of this piece of epiblastic tissue giving rise ultimately to the formation of a dermoid cyst. Such a cyst has a wall of skin and contents of thick, turbid material containing desquam-

ated cells. In this situation such tumours do not, as a rule, contain complex structures. Superficially a dermoid tumour appears as a rounded, definitely limited tumour, adherent to deep structures, but with the skin movable over it.

In this case the X-ray photographs show the outer edge of the tumour as a ring of bone with a clear centre. The radiograms also show with accuracy the position and extent of the growth. In its growth the tumour, by filling up the orbital cavity, caused a gradual protrusion of the eyeball, and when the pressure was removed the eyeball regained its normal position.

Treatment.—In such cases of dermoid cysts removal is the best treatment. In this instance the patient was put on a course of potassium iodide, but no improvement was observed. Operation was then performed and the cyst obliterated. The sac must be carefully dissected out, or if this is not possible, it should be cauterised, care being taken that no portion is left behind, otherwise recurrence will follow.

Case III.—A Tumour Arising from the Coccygeal Gland.

Henry H., aged 22 years. Admitted to Ward 10, 24th November 1915. Complaint, "pain in the left leg."

History.—Patient said that about eleven weeks previous to admission (August 1915) he first experienced a severe pain in the small of the back which prevented his moving about, especially interfering with the movements of bending and straightening the back. A week or so later the pain began to pass down into the lower extremity. It was a shooting pain and was very severe, but it was intermittent in character. At first it appeared in the top of the thigh, especially in the gluteal region. There was never any pain in the inguinal region. The pain later shot right down the back of the thigh to the knee, and later still to the heel and along the sole of the foot. He never had pain in the front of the thigh or leg. The pain occasionally shot along the inner side of the foot. There never was pain in the right leg or over the front of the abdomen. The patient said that his left foot was somewhat weak, but had never noticed any weakness of the thigh or leg. Sometimes he complained of a sensation of "pins and needles" in the foot. There were no alimentary symptoms, and no pain or difficulty in defæcation. He said, however, he had lost about 2 stones in weight.

Family History.—Father alive and well; mother alive and

well; six brothers and three sisters, all well. He was hitherto a healthy man, and had had no venereal disease. He took alcohol to a moderate extent. His pulse was 80 per minute on admission, and temperature 98·2° F. There was a mitral murmur present in the heart, but the lungs appeared healthy. Appetite was good. Bowels were regular. Sleep was bad at times.

Examination of the Patient.—Left thigh and leg were somewhat wasted. There was no impairment of sensation to touch, pain, heat, or cold. Left knee-jerk was slightly more active than the right. Superficial reflexes were active. The plantar reflex was flexor. No clonus present. There was weakness of the extensors of the foot and flexors of the knee of the left lower extremity. Flexors of the hip were weak on both sides. There was no swelling visible in the region of the sacrum.

Rectal examination disclosed a tumour in the posterior wall fixed to the sacrum—hard, smooth, and immovable. The finger could not be passed beyond it, and the lower border was about 1½ to 2 ins. above the anus. There were no palpable glands.

X-ray Examination.—The shadow of the sacrum and coccyx was almost obliterated, but there was not any shadow apparently superimposed. The report stated that it was seldom that this effect was produced by sarcoma. It was common where there was an epithelial tumour (malignant) in the vicinity of the bone.

The patient was under observation for a few days, and operation was decided on.

Treatment.—Operation, 30th November 1915, *by Mr. Miles.*—The anæsthetic used was chloroform, followed by ether.

An incision was made in the mesial plane over the sacrum from the 3rd sacral segment down to within half an inch of the anal orifice. The coccyx and lower portion of the sacrum were laid bare by dissection. The muscles and ligaments attached to the lower portion of the 4th to 5th sacral segments and coccyx were divided on either side, and the extent of the tumour ascertained. It was felt to extend up as far as the 1st sacral segment. The tumour was dissected out of its bed, but as it was attached to the two lower sacral segments and coccyx these segments were removed along with the tumour. The bleeding-points were ligated, but there was considerable oozing, especially from the divided bone. Hot saline was applied, and then deep sutures were put in; the central part of the wound was packed with iodoform gauze. The wound was stitched on either side of the drainage opening. The rectum was not involved, and was not

interfered with. A ¼ gr. morphia suppository was inserted into the rectum.

Progress Notes.—For two days following the operation patient was unable to pass urine, and the passage of a rubber catheter caused great pain. He was put on small doses of hexamine to prevent the occurrence of cystitis. At the end of this time patient was able to pass water himself, and felt more comfortable.

4th December 1915.—The packing was removed from the wound and replaced by red lotion. Eusol and red lotion dressings were applied alternately. No discharge from the wound.

On 9th December the stitches were removed. There were no complications, either local or general, and the temperature remained steady.

10th December.—Patient got up.

27th December.—A rise of temperature set in. There was nothing septic about the wound. There was a slight rash noticeable on the buttocks.

4th January 1916.—Temperature was now settled again.

10th January.—Patient again got up.

22nd January.—Progress good. Wound was dry. Went home. Still some slight weakness of the extensors of the toes, and pain in outer side of the foot. The patient at this time said he felt in the best of health. He was of good colour, had gained weight, and he was confident of prolonged good health. The next that was heard of him was when he returned to Ward 10, on 1st March 1916, on account of the recurrence of pains in the left leg.

Re-admitted to Ward 10, 1*st March* 1916.—Complaint, recurrence of pains in the left leg.

Further History.—Patient said that for the first fortnight after leaving hospital he enjoyed good health, though at times there was some stiffness in walking with the left foot. There was no definite pain at this time. About the beginning of the second week in February 1916 he began to have attacks of pain in the back of the left thigh. These pains came on at irregular intervals, and interfered with his sleep. Some nights he was free from pain. He said that pressure over the back of the thigh relieved the pain, and this condition of affairs persisted up to the time of re-admission. The left foot then started to trouble him. About the middle of February he began to have pains of a "pins and needles" character along the outer border of the foot. The

foot began to swell on its dorsal aspect, and his toes became stiff and difficult to move. The swelling persisted for a couple of days, and was not of an inflammatory nature. He was also troubled with pains in the medial aspect of the thigh and affecting the left testicle and cord. The right leg was unaffected. He had no trouble with defæcation, but he was occasionally troubled with constipation.

Examination.—Left leg and thigh not so well developed as the right, and the muscles were flabby. There was marked foot-drop present, and pain was complained of over the outer side of the thigh from the knee to the hip, more marked at one time than another.

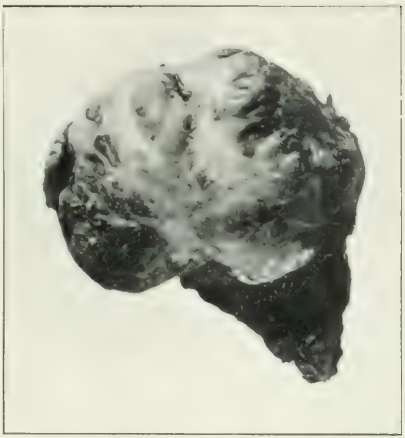
Rectal Examination.—This revealed a mass in the posterior wall of the rectum near its lower end on the right side. There was a much larger mass higher up on the left side, attached posteriorly to the sacrum, and immovable. The finger could not be passed beyond it. The rectum was loaded with fæces.

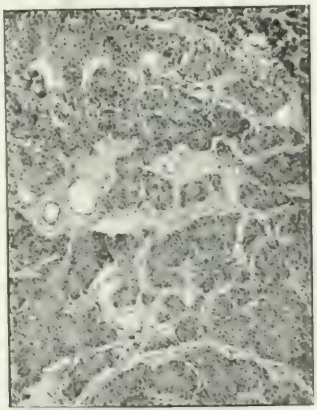
Treatment.—The growth was too extensive to make operative treatment advisable, and palliative treatment was resorted to. At times the patient was in great pain, chiefly referred to the leg and foot; rolling about in bed, and sweating profusely. During these exacerbations of pain the administration of an enema clearing out the bowel was invariably followed by marked relief of the symptoms for a time. When the pain was severe, heroin in doses of $\frac{1}{12}$th gr. was given hypodermically as required. Belladonna and glycerine fomentations were applied to the leg and foot, and were found to be very soothing. A splint was applied to support the foot, and as symptoms had abated he was sent home on the 11th March 1916, and his doctor advised of the condition of affairs.

Pathology.—The tumour removed at operation was preserved, and a microscopic examination of the tissues was made.

Naked-Eye Appearances.—The tumour mass was about the size of a fœtal head, but somewhat flattened above (Fig. 4). It was intimately attached to the lower part of the sacrum and coccyx. The tumour was smooth on all sides except superiorly, where the tissues had been cut through, and was very vascular at the time of the operation. There were no adhesions anteriorly. It was fairly firm to the touch, but not hard.

Microscopic Appearances.—(Fig. 5.) It was seen to be a growth of a malignant nature, and the cells were arranged in masses, and in some areas in irregular columns. In other areas of the section





the cells had become detached and more isolated. Under the high power the cells were seen to be large polyhedral cells, with well-staining nuclei and definite staining granular protoplasm. There was a marked presence of chromatin material in the cells, and mitosis was present. The stroma was scanty, but was of well-formed fibrous tissue. The blood-vessels were numerous, but not well supported, and the walls were thin, some consisting of badly-supported endothelium. In certain areas hæmosiderin pigment was seen among the stroma and cells.

It would appear, therefore, from the position of the tumour and the variety of cells, that it was derived from the coccygeal gland and of the nature of an adeno-carcinoma, the term adeno-carcinoma being used in the sense that it is a tumour containing epithelium grouped both in gland form and in solid masses.

Little is known about the functions or pathology of this coccygeal gland, but its histology has been investigated by some workers. It was first described by Luschka in 1860. It is a small body, about the size of a pea, situated just above the attachments of the sphincter ani to the coccyx. It is supplied with blood from the middle sacral artery, and is said to have a sympathetic nerve supply.

Histologically it consists of numerous loops of blood-vessels and sinuses with free communication, and surrounded with several layers of granular polyhedral cells containing a hyperchromatic nucleus. These cellular areas are intersected by bands of dense connective tissue.

It is of more than ordinary interest to find a tumour of such rapid growth and powers of spread arising from the coccygeal gland. Tumours have been met with in this region, but of the nature of teratomas. Hutchinson and others describe cases of tumours arising in the coccygeal gland, but congenital in origin, occurring in young children and giving rise to a very prominent external swelling.

In this particular case there is no evidence of an hereditary factor to explain its occurrence, and it appears to have been of rapid growth.

Clinical Features.—The main symptom that this patient complained of on admission was pain in the left thigh and leg. This pain was intermittent in character, severe, and often worse at night. The course of the pain was first in the gluteal region, then down the back of the thigh, and later down to the foot. These features pointed to a neuralgic condition of the sciatic

nerve. As regards the diagnosis, was the sciatica a primary condition, or was it secondary to some affection of the pelvis or cord? As is well known, pain referred to the sciatic nerve is a symptom of pressure upon the nerve from tumours, aneurisms, etc., within the pelvis. The possibilities that could be met with in this case were—

1. *Primary.*—Inflammatory condition of the nerve as seen in gout, chill, etc.

2. *Secondary.*—(*a*) Pelvic tumours causing pressure on the extra pelvic portion of the nerve. (*b*) Pelvic tumours causing pressure upon the nerve within the pelvis, *e.g.* sarcoma of the pelvic bones; rectal cancer. (*c*) Diseases of the hip-joint; arthritis deformans; tuberculous disease. (*d*) Tabes.

Of the secondary conditions the possibilities that most readily suggested themselves were intrapelvic tumours. A digital rectal examination was made, and a tumour mass was found fixed to the sacrum, smooth, hard, and immovable. The next question was, What was the tumour? was it rectal cancer or was it pelvic sarcoma? It was not carcinoma of the rectum, for there was no invasion of the mucous membrane of the rectum by tumour growth or ulceration. There was no undue pain complained of on rectal examination. There was no history of pain on defæcation, blood in the stools or passage of blood-stained mucus as would be met with in typical cases of rectal cancer. The diagnosis appeared to be, then, sarcoma of the sacrum. (1) There was the presence of a tumour connected with the sacrum. (2) Rapidity of growth of the tumour. (3) Age of the patient. (4) Loss in weight—2 stones in about 3 months. To make the diagnosis more certain an X-ray examination was made, and this pointed to a carcinomatous rather than a sarcomatous growth. At the operation the tumour was found attached to the lower sacral segments and coccyx, but not involving the posterior rectal wall. In removing the tumour it seems probable that pieces of this tissue still remained from the upper cut surface of the tumour mass. As a result of operation the patient got relief from his sciatica-like pains, but there was still a certain amount of weakness in the leg. The cessation of pain was due to the removal of the mass pressing upon the roots of the sciatic nerve. The operation was performed on the 30th November 1915, and the patient left hospital on 22nd January 1916. On 1st March he was back to the ward on account of the great pain in the left leg and the drop-foot that had developed. Examination now showed the presence of two large tumour masses

in the pelvis. Recurrence had therefore occurred to this extent in 3 months. The increased severity of the pain was due to the greater pressure within the pelvis upon the sacral nerve roots. Paroxysms of pain came on at times, and it was then found that the rectum was loaded with faeces. This increased the intra-pelvic pressure, and also no doubt produced pelvic congestion by impeding the return of blood from the veins. The pain complained of in the medial aspect of the thigh appears to have been due to pressure upon the obturator nerve roots. The foot-drop and muscular weakness that this patient exhibited was due to the long-continued pressure upon the sacral nerve roots. The degenerative effect upon the nerves is shown by the degeneration in the external popliteal nerve, where there was weakness of the extensor muscles of the toes and affecting the peronei and tibialis anticus muscles, resulting in the production of "foot-drop" and the characteristic steppage gait that this patient exhibited when walking.

In these phenomena we see the effect of prolonged compression upon a nerve. In the initial stages the signs are those of irritation—cramps and neuralgic pains. In later cases, where the compression has been prolonged, the symptoms pass into those of paralysis, sometimes associated with trophic phenomena. As a result of operation the pressure of the tumour was relieved for a time, and the effects of recovery in the nerves was shown by the disappearance of the paroxysmal neuralgic pains and a better degree of muscle tone and power. Complete recovery in the nerves and muscles, however, did not occur, as there was weakness of the foot persisting.

Case IV.—Abscess in the Femur Due to Infection with Bacillus Coli and Bacillus Paratyphosus.

A. M., aged 33 years; Sergeant-Major 1 1st ———. Date of admission, 16th November 1915. Complaint, pain in the left leg.

History.—Date of onset, 23rd October 1915. Patient was out in the trenches in Gallipoli, and one day while under shell fire he got a blow on the left thigh from a stone or fragment of shell. This caused him pain and stiffness in walking for some time. A day or two later he began to feel "seedy and out of sorts," but he did not report sick at this time. The medical officer, however, noticed that he was not well and took him off

duty. He felt fevered (his temperature, he says, was 104·6° F.) and had severe pain in the left thigh. The pain was gnawing in character, and boring into the bone. The thigh was beginning to swell and was very tender. He was conveyed by ambulance to the base and then transferred to a hospital ship. He was confined to bed and was suffering greatly from the pain in the thigh, which appeared to be spreading in extent. The thigh was painted frequently with iodine. While on the homeward voyage he contracted pleurisy. Since admission the extreme pain had passed off, but there was still some pain, worse at one time than another, and there was tenderness over the swollen area. Patient said that he used to sweat a good deal at night, but that has now stopped.

Examination.—Left thigh was swollen. There was a fusiform swelling anteriorly in the line of the upper and middle thirds of the femur and extending laterally. There was redness of the skin on the lateral aspect of the thigh extending around the region of the great trochanter. The muscles below this swollen area were wasted. Palpation showed a degree of extreme tenderness at a point in the line of the femur at the junction of the upper and middle thirds, and pain on pressure 2 ins. below the great trochanter.

Pulse, 108; respirations, 20; temperature, 101·8° F. He had always been a healthy man and had no previous illnesses. There was nothing of interest in the family history.

Operation, 29th February 1916, *by Mr. Miles.*—An incision was made over the shaft of the femur on the outer side just below the trochanter parallel to the shaft. The bone was exposed and the shell over the abscess gouged away. Pus lay in the tissues around the bone. The cavity was found to contain pus, and this was evacuated. The inner wall of the cavity was then scraped freely and a cloaca found to pass forward to the anterior surface of the bone. The cavity was packed with eusol swabs. Anæsthetic used was chloroform, followed by ether.

(1) The pus from the tissues round the bone and (2) the pus from the abscess cavity in the bone were examined by the bacteriologist.

In the former, from examination of films and culture the organism present was the bacillus coli. In the pus from the abscess cavity, by a similar examination the organism appeared to be the bacillus paratyphosus.

Treatment (*Post-Operative*).—The bone cavity was packed with

swabs soaked in eusol. When the dressings were changed eusol was syringed through the abscess cavity and then packed with eusol swabs covered with waterproof, wool, etc. An abscess was found pointing near the great trochanter. This was opened, and there was a communication between this abscess cavity and the bone cavity. Eusol was syringed through the two.

Commentary.—Diagnosis.—The chief difficulty in this case was that of a correct diagnosis. Initially the patient was suffering from pain in the left thigh, swelling in the region of the joint, with impairment of, and pain on, movement of the limb. There was also a high temperature. At this stage, before symptoms became more definitely localised, the diagnosis seemed to lie between—

(a) *Acute Rheumatism.*—On admission to the Infirmary he was considered a medical case and was put on anti-rheumatic treatment. There was no relief in the symptoms, and the temperature remained elevated at 100 to 103 F. This form of treatment was suspended. Sweatings at night and the development of a swinging temperature gave rise to the possibility of the condition being of (*b*) a *tuberculous nature*. He was put on cod-liver oil and creosote. There was not much effect at first, but in a week or two the temperature became more settled, though the evening rise and morning fall was still evident. During this time the local symptoms in the left thigh were present. There was swelling of the upper part of the thigh, some redness of the skin, and pain which the patient felt to be localised in the bone itself. This pain was often severe at night and interfered with his sleep. The condition appeared to be more a surgical than a medical one. The patient was X-rayed, and Mr. Miles was asked to see him. The result was that operation was performed and the condition found to be an abscess in the upper part of the femur. This last point, however, was not easy to clear up before operation, for among other conditions (tubercle, syphilis) that had to be considered was the question of malignant disease, viz. sarcoma. In such cases of subacute and chronic inflammatory disease of bone with localised swelling the differential diagnosis of sarcoma is not easy. In the case of a chronic inflammatory condition the most frequent lesion is a Brodie's abscess resulting from a previous acute pyogenic osteomyelitis. In this case there was no such history to help us. The point was inquired into particularly, and there were no

scars or marks present on the limb that could point to healed sinuses.

(c) *Was it Sarcoma?*

1. *Age.*—Sarcoma of bone is commonly met with in people up to 30 years of age. As this patient is only 33 years of age this point was of no value as an eliminating factor.

2. *Pain.*—In cases of central sarcoma this appears early in the disease. It is a constant, aching pain, worse at night, but not increased on movement. In this case pain was severe, intermittent at times, worse on movement and on pressure, and definitely localised at one point.

3. *Swelling.*—In periosteal tumours swelling is an early symptom but a later symptom in central sarcoma. The swelling is fusiform—"leg of mutton" shape, localised, and with a definite contour. The consistency varies in different parts, and usually there is a crackling sensation on deep pressure. In central sarcoma a pulsatile tumour may be felt. In this case swelling appeared fairly early, and with the signs of an inflammatory reaction—pain, heat, swelling, redness, constitutional disturbance, and interference with function.

4. *X-Ray Appearances.*—This is the best and one of the most reliable points in making a diagnosis. In sarcoma an ossifying tumour is easily recognised by the appearance of the new-formed shell of bone. In central sarcoma, which is cellular, the bone shadow in the tumour area is obliterated and the adjacent bone of the shaft is of normal appearance.

In this case the X-ray picture showed a well-marked circumscribed area in the upper part of the shaft of the femur, with thickening and sclerosis of the surrounding bone. From a consideration of these various points it will be seen that most information is obtained from the X-ray appearances, and this is true, generally, of bone lesions, for not only is the nature of the lesion made manifest, but its localisation is rendered accurate.

How did this Abscess in the Femur Arise?—Examination of the pus obtained at operation by the bacteriologist showed the presence of bacillus coli and bacillus paratyphosus.

The patient was in Gallipoli some months previous to his illness. Where so much typhoid, paratyphoid, and dysentery abounded it is only reasonable to suppose that this man's intestinal tract contained a considerable number of organisms of the coli-typhoid group, but not producing symptoms. As Kocher and Sahli have pointed out, the blood-stream can become

infected with micro-organisms from the intestinal tract and set up a pathological process when the tissues are rendered favourable by diminished resistant power. This latter element is present in this case, where we have trauma producing this state of affairs.

The man sustained a blow on the thigh, severe enough to cause him pain and discomfort on walking. A traumatic periostitis may have been set up in this part of the femur, leading to a lowering of the vitality of the bone in that area. The organisms circulating in the blood-stream would readily reach this favourable part by reason of the rich vascular supply found in the end of the long bones. The bone marrow in this area was not in a condition to resist the organisms, and they gained the upper hand. This lowered vitality of the marrow, however, was only localised, and the healthier marrow was able to prevent the spread of suppuration. The general signs of the inflammatory reaction were present. As time went on this localised area of suppuration set up an irritative condition of the surrounding marrow, causing stimulation of the osteoblasts with the resultant production of new bone, giving rise to sclerosis of the shaft round the abscess cavity. The periosteum by laying down new bone on the surface in this area gave rise to thickening of the shaft—hyperostosis.

Case V.—Nephritis Treated by Double Decapsulation of the Kidneys.

J. J., aged 6 years. Admitted to Ward 9 on 9th February 1916. Complaint, swelling of the body and eyes.

History.—In October 1915 she was admitted to Ward 33 under Sir Robert Philip, on account of vomiting and swelling of the body and face. The facts obtained then were as follows:—

On the Tuesday three weeks before admission to Ward 33 the child took ill while she was at school. When she got home she took a fit of shivering and vomited, and her nose bled a little. A week later she vomited again. She complained of having a sore belly in the right side when she coughed, and also that her back was sore. On the first Saturday her eyes swelled somewhat and she was passing very little urine. On the Sunday her body was swollen; this continued till the following Thursday, when she had the second attack of vomiting and felt worse again. After the Tuesday the doctor gave her some medicine which increased the quantity of urine; it had a very solid deposit on standing, and

was of a reddish colour. She had no rash, nor had any of the children in the house, though there were cases of scarlet fever in the next house.

Examination on Admission to Ward 33.—Child was somewhat swollen about the body and lower limbs, but looked fairly well. The following morning she developed a rash and slight temperature. The rash was on the anterior aspect of the right thigh, and was of a homogeneous rosy red colour and not raised; very tender to the touch. Next morning the rash had faded, but appeared in the posterior aspect, but this also disappeared. There was no sore throat, and no signs of desquamation. Swelling gradually disappeared, only a little remaining in the legs. There were 2 grs. albumen to the ounce, and 10 ozs. urine were passed at first. She was kept on milk diet with an occasional rusk. Hot baths were also given. She was under medical treatment until 9th February 1916. During this interval the dropsy continued to develop, but the increase in the œdema was not constant, at times the child being less puffy than at others. The secretion of urine fluctuated; on the average the daily urine passed was 20 to 25 ozs. in the 24 hours. The highest on two occasions was 40 ozs., but on various occasions the amount passed was as low as 10 ozs. The urine contained a considerable amount of albumen; at times the albumen would rise to 12 to 16 grs. to the ounce of urine, and again would remain at 1 to 3 grs. per ounce for some days. The excretion of urea also fluctuated.

The patient was transferred to Ward 9 on 9th February 1916. At that time the patient was very dropsical. The face was pale, very puffy, and the eyes occluded by the œdematous lids. The limbs were very œdematous also, and the peritoneal cavity contained fluid. The vulva was also distended with œdema. Respiration was embarrassed, and the apex-beat was displaced to the left side. The albumen in the urine was 6 grs. to the ounce, and microscopically there were hyaline and granular casts. Operation was decided on. Both kidneys were to be decapsulated at the one operation.

Operation, 11th February 1916, *by Mr. Miles.*—Anæsthetic, open ether throughout. The right kidney was exposed first by an incision about one inch below the last rib and parallel to it, extending from the outer border of the erector spinæ muscle. This was carried down to the level of the muscles of the abdominal wall, and the fascia behind the external oblique was opened into. The posterior fibres of this muscle were cut and the perirenal

fascia divided and the kidney drawn into the wound. It was large and congested, with one or two paler areas. The capsule was incised in its long axis and stripped off almost to the hilum and then clipped away. The kidney was replaced and the wound closed. The left kidney was similarly dealt with, and the child returned to bed. During the operation the extremities were wrapped in cotton-wool and a large pad of wool placed on the chest. The whole operation lasted about 16½ minutes, and the child rapidly recovered from the anæsthetic and was able to understand what was said and to speak in reply in about 10 minutes. There was practically no vomiting.

Progress Notes.—13*th February* 1916.—Wounds healthy.

21*st February.*—Stitches out.

25*th March.*—Got up.

8*th April.*—Went home.

Commentary.—The points that require especial notice in connection with this case are: (1) Effects of the operation on the urine and œdema. (2) The operation — indications for and against. (3) The anæsthetic.

The Urine.—In October 1915, when the child fell ill, the symptoms were shivering, vomiting, pain in the back, swelling of the body and eyes, and marked decrease in the amount of urine voided. The urine passed was high-coloured, and had a thick, solid deposit. The girl had contracted an attack of acute nephritis, such as would follow a chill or scarlatina. In such a condition the characters of the urine are—

Quantity.—Diminished at times to a great degree. If the acute stage tends to pass off, the quantity increases.

Colour.—Dark. Clears up as the acute stage passes off.

Sediment.—Copious and brownish in colour, due to urates, blood, and colour matter.

Urea.—Diminished in amount.

Albumen.—Is large in amount.

Casts.—Hyaline and granular casts in considerable numbers, also red blood-cells.

A daily examination of the urine was made, and a record of the amount passed was kept. These figures have been incorporated in the appended tracing of the curves (Fig. 6). One curve represents the amount of urine passed in ounces per day. The second curve represents the quantity of albumen present, measured in grains per ounce. These records cover the time from admission to the Infirmary until a few days before discharge. These curves

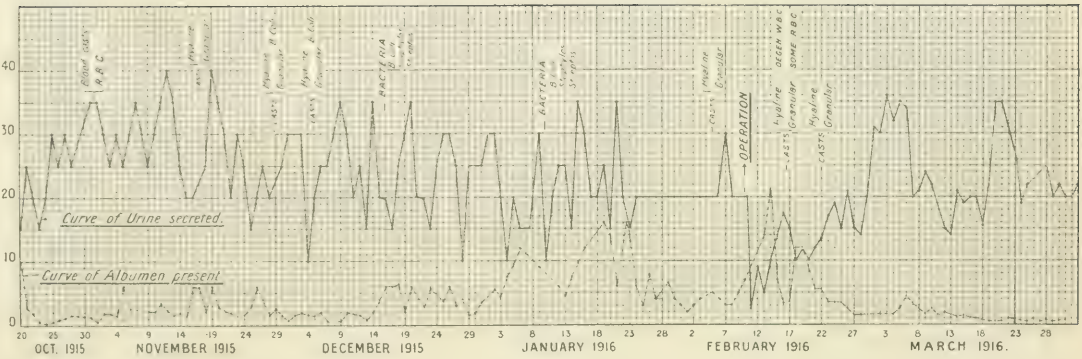

Curve of Urine secreted.
Curve of Albumen present
OPERATION
BACTERIA
OCT. 1915
NOVEMBER 1915
DECEMBER 1915
JANUARY 1916
FEBRUARY 1916
MARCH 1916.

Fig. 6.

illustrate the fluctuation in the amount of urine, and of the amount of albumen from day to day. The effects of the operation are also to be noted on the two curves.

During the three and a half months the patient was under medical treatment dropsy persisted; the amount of urine varied from 10 to 40 ozs. per day, and the albumen from 0·3 gr. to 16 grs. It seems, therefore, that the acute condition had passed into a subacute or chronic condition. A chronic diffuse nephritis is often the sequel to an acute nephritis, especially those cases from scarlet fever. In such a condition of the kidneys the characters of the urine are—

Quantity.—Diminished in amount but not so great as in acute forms, and the daily amount fluctuates more markedly.

Albumen.—This is always present, and may be in large amounts which are subject to great variations.

Casts.—Are numerous. In earlier stages hyaline and granular predominate. Red blood-cells are present in acute exacerbations.

It will be noticed from the curves of urine and albumen that on the day following operation there was almost complete suppression of urine, only 2½ ozs. being voided, and on heating become almost solid from the great amount of albumen present. On the following day the amount passed was 8½ ozs., and thereafter the amount of urine showed a tendency to increase. About three weeks after operation the amount of urine rose quickly to 30 to 35 ozs. and remained at this for about a week, and then fell to 20 ozs. and thereafter averaged 25 ozs.

As regards the albumen for the first week after operation the quantity varied much, but after that it began a fairly steady and constant decrease, and for the month before discharge varied from 0·313 gr. to 0·619 gr. per ounce.

As regards the oedema there was no perceptible effect until about a fortnight after operation. When the urine became copious the dropsy began to disappear more quickly, and finally disappeared. When able to get up the child was very lively and in good health, and nothing took place to interfere with her convalescence. When she was discharged there was still a slight amount of albumen present in the urine.

Operation.—The operative treatment of nephritis is of fairly recent date. The first mention of surgical interference in kidney disease was in 1897, when Harrison proposed puncturing the kidney for cases of hæmaturia with albumen and renal pain. He found that by this method, and later by incising the capsule,

these symptoms cleared up, probably from the relief of congestion in the affected kidneys. In 1899 Ferguson claimed that two of his cases of chronic interstitial nephritis had been cured of symptoms by incision of the capsule and renal puncture. It was Edebohls in the same year who advocated the operation of decapsulation of the kidneys in Bright's disease, and in 1901 recorded a series of cases successfully treated by this means.

How is Relief Brought About?—The kidney is enclosed in a capsule of very slight degree of elasticity, and tension is quickly produced by engorgement from the increase of blood in inflammation, by œdema, and by proliferation of the kidney elements. In such a case, then, incision or stripping off this capsule will relieve this tension, and give the kidney a better chance to recover. Edebohls' hypothesis was that in decapsulation relief was due to the formation of a new circulation through the new-formed adhesions: these adhesions contained more arteries than veins; that the free blood-supply absorbs exudate and products of inflammation; frees the tubes and glomeruli from pressure and constriction; causes the re-establishment of a normal circulation and the regeneration of epithelium.

It is not easy to see, however, how this increased blood-supply comes about. When the raw surface of the kidney is replaced in its bed adhesions are sure to form, and in the natural process of repair scar tissue will result which is usually devoid of blood-vessels.

The exact status of this operation has not yet been definitely fixed, but there are cases in which operation is contra-indicated, and cases in which it is called for. Operation, however, should not be resorted to unless medical treatment has been tried and proved unavailing, and where the symptoms are seen to be getting more aggravated and even dangerous.

Indications for Operation.—(1) Persistent dropsy; (2) uræmia; (3) excessive albumen; (4) suppression or persistent and notable diminution of the amount of urine voided.

The type of cases are acute nephritis or chronic nephritis with acute exacerbations. The best results appear to occur in the chronic parenchymatous nephritis in which there is marked anasarca.

In this particular case operation was justified on the following points:—(1) Failure of a long course of medical treatment; (2) marked dropsy; (3) high amount of albumen; (4) diminution in the urine voided; (5) age of the patient.

Operation is contra-indicated in (1) cardio-vascular disease; (2) patients over 50 years of age. As in this case both kidneys may be done simultaneously, or they may be done on two separate occasions.

The results of the operation on the girl are in keeping with those found in general in such operations—(1) Disappearance of dropsy; (2) increased secretion of urine; (3) great diminution in albumen. After operation there is transient anuria; then in about 10 to 14 days polyuria sets in. In about a month there are decided signs of improvement, and improvement is gradual. These points are illustrated in this particular case. Whether permanent cure is established is doubtful, but there is great amelioration of the symptoms.

The Anæsthetic.—The anæsthetic used in this operation was ether. The child went under quietly, and deep anæsthesia was obtained in about 7 minutes. The ether was administered by means of an ordinary mask covered with gauze, and a fold of gauze placed over the patient's mouth and nose. During the operation the body had to be turned on one side, but no respiratory difficulty was met with despite the great amount of œdema. All parts of the patient outside the area of operation were swathed in cotton-wool to counteract the cooling effects on the body of ether, and to lessen the shock. When once deep anæsthesia was obtained very little ether was given, and the operation was performed rapidly. The child on being taken back to bed soon recovered from the effects of the anæsthetic. There was practically no vomiting. There was no disturbance of the cardiac or respiratory systems.

There is considerable difference of opinion as regards the choice between chloroform and ether in such cases. Both forms of anæsthetic are said to produce renal irritation with albuminuria, but chloroform is more liable to produce albuminuria in supposed healthy kidneys, and ether is said to increase a pre-existing albuminuria as well as hæmaturia, and a tendency to suppression of urine. These effects of the ether, however, can to a great extent be eliminated by careful administration, and when such precautions are taken renal complications are exceptional. Again, in regard to the question of shock, patients undergoing operations on the kidneys, and more especially when both kidneys are being dealt with, are particularly liable to shock. Again, from the nature of the operation the position of the patient interferes with free breathing. Anæsthesia must be deep, in order that the

surgeon may have proper access to the kidneys. Taking these points into consideration ether is to be preferred to chloroform, provided that due care is taken in its administration, and no excess of the anæsthetic is given.

The cases were all treated in the surgical wards of the Royal Infirmary under the charge of Mr. Miles, to whom I am indebted for permission to publish this record.

A CASE OF PROGRESSIVE SUBCUTANEOUS OEDEMA.

By D. M. GREIG, C.M., F.R.C.S.(Edin.),
Senior Surgeon, Dundee Royal Infirmary

THE subject of this variety of chronic oedema of the lower extremities was an unmarried nulliparous female. She was an illegitimate child, and her family history was not obtainable. Though able to earn her livelihood as a millworker she was without doubt mentally deficient. She would be correctly described as feeble-minded—"a person capable of earning her livelihood under favourable circumstances but incapable of competing on equal terms with her normal fellows." She was happy and good-natured, though childish and without ambition. She could both read and write, but was facile and easily pleased. Her cerebral condition, however, had apparently no relation to her physical abnormality. She had had the usual illnesses incidental to childhood, but no other of any consequence. She was well-nourished and well-developed, and of average stature. She was inclined to stoutness, but the deposition of fat appeared to be equally distributed throughout the body. When 14 years of age she went to Brisbane, Queensland, where she remained for over a year. Soon after her arrival there she had a mosquito bite on the left leg. It suppurated and discharged, and the site of the wound became the site of a chronic dermatitis. It was in consequence of this condition that she was sent home, and on her arrival had an open sore on the left calf the size of a crown piece. One or two other points round the principal ulcer were open and discharging. Though under treatment these gradually healed, the limb remained somewhat swollen, and this swelling, though originally limited to the area of ulceration and below it, gradually spread upwards until the whole limb became involved. It had continued during ten years before the right leg became affected, and except when she was under treatment the swelling had steadily progressed. She died at the age of 32, but I have been unable to ascertain the exact cause of death, though the onset of the illness appears to have been started by an attack of erysipelas in the left limb.

She first came under my observation four years before her death. The enlargement of the limbs was very obvious, and most distinct in the calves. The circumference of the left calf was

25 and the right calf 17 inches. The feet, probably from the support afforded by her boots, were but slightly affected, and appeared absurdly small in comparison with her enormous legs. The site of the originating insect bite was marked by a crateriform depression surrounded by a raised firm margin, quite an inch broad (Fig. 1). This ring of induration was pinkish in colour, and marked by several deep grooves which radiated from the central depression outside the pink area. There was a tendency to heaping up of epithelium or warty formation. The whole overgrowth was firm in consistency, and neither painful nor tender. Each leg formed an enormous fusiform swelling crossed by several deep transverse lines (Fig. 2) There were several raised nodules about the skin, but the general appearance of the cutaneous surface was healthy. She complained of the weight of the limbs and that they got very easily tired on exertion, and she believed that the condition had increased considerably since she started work as a millworker four years ago. There was no glandular enlargement in either groin. The abdomen was full, but the abdominal wall did not participate in the œdema, and there was nothing abnormal apparently in the viscera. There was no venous engorgement. The feet had a tendency to be cold, and were often slightly bluish. She had been under treatment in various hospitals, but the treatment had consisted only of rest in bed and the application of pressure by bandages. Each course of treatment was followed by considerable reduction in the size of the limbs as long as she was in bed, but the swelling returned despite bandaging as soon as she resumed the erect posture. In view of the incomplete and temporary nature of the improvement effected by such methods I concluded that some more drastic method should be employed, and therefore did a lymphangioplasty in the left limb, introducing long strands of sterilised thick silk from the ankle to the groin on the outer and inner side of the limb, and from the ankle to the gluteal fold posteriorly. Six months later the left limb was not so swollen and did not feel so tense, and the measurement round the calf was four inches less than before the lymphangioplasty. The subjective symptoms, however, of weight and tiredness on exertion were unrelieved, and altogether the paltry success on the left limb did not invite a similar procedure being carried out on the right.

The term progressive subcutaneous œdema describes accurately the principal features in the class of cases to which the above belongs. The general appearance of the affected parts and the

characters noted in a cursory examination might lead one to believe that there were extensive and gross changes in the subcutaneous tissue itself or even in the skin, but the paramount part which œdema alone plays in the production of the condition is evidenced by the enormous improvement which follows prolonged recumbency and the application of pressure. Changes in subcutaneous tissue are certainly not absent, but they are not of such primary importance as to justify the description of such cases as "chronic subcutaneous fibrosis." This name Stockman introduced to describe a condition of œdematous overgrowth associated with irregular and extensive distribution of fat in the subcutaneous tissues. He ascribes it to chronic irritation of the fibrous tissue caused by such general affections as rheumatism, influenza, general gonococcal infection, mucous colitis, and "other imperfectly recognised causes."[1] He points out that the small blood-vessels show periarteritis and endarteritis and that there is an interstitial inflammation of the small nerve twigs, "the whole being evidently the result of a reaction of local irritation by microbic toxins." In such generalised affections it is to be expected that the alterations would be general throughout the body, and this Stockman maintains, but though my patient was inclined to slight obesity, there was nothing abnormal in the panniculus adiposus throughout the body, only in the affected limbs.

That the originating cause was septic inflammation scarcely admits of a doubt. The sore, started by an insect bite, became the site of chronic inflammation, and remained open and discharging for more than a year. From this sore was doubtless set up a lymphatic irritation, microbic in origin; a progressive lymphangitis, with obstruction of the lymphatic channels. It was neither of the nature nor of the acuteness to cause a lymphadenitis, and had the peculiarity of progressing locally until, having affected the entire limb, it proceeded to affect the lymphatic channels draining the opposite one. The tissues affected are more or less altered, the fat becomes firm, the fibrous septa thicker, and the skin altered in texture and condition. Without doubt in these structures a periarteritis will commonly be found, but there is no clinical evidence that an endarteritis exists. The condition is totally different from the swollen extremity where enteric, puerperal, or other affection has produced a venous thrombosis. In those, as in chronic lymphangitis, relief is obtained by rest, but the swelling is much more regular, the parts of the limb retain their proportions and are not distorted as in lymphatic œdema,

and the condition, though extensive, is not progressive from limb to limb.

In progressive subcutaneous œdema the primary change produced by the absorption of septic toxins must be looked for in an endolymphangitis, and this change in the epithelium is centripetal. There is no perilymphangitis in the initial changes. That is entirely due to the secondary changes which take place in the tissues of the affected parts generally, including those around the lymphatic vessels. There is no lymphadenitis. Indeed it is possible that an actual atrophy of the lymphatic glands may result from the condition, for the afferent and efferent channels are altered and the function of the glands interfered with. It is in the vessels themselves and not in the glands that changes must be looked for, and it is by the vessels that the progression takes place until the opposite limb, and by-and-by the trunk participates in the condition.

It is the ideal condition for lymphangioplasty. Lymphangioplasty replaces the obstructed lymphatic ducts by channels along which, by capillary attraction, the serous effusion is drawn from affected to healthy subcutaneous tissue, where it may be reabsorbed in the natural way. One cannot hope for more than temporary alleviation by rest and bandages, but a lymphangioplasty may provide a scientific, if crude, replacement of the obstructed vessels.

Reference.—[1] Stockman, R., "The Clinical Symptoms and Treatment of Chronic Subcutaneous Fibrosis," *Brit. Med. Journ.*, 18th February 1911.

OBITUARY.

WILLIAM TAYLOR, M.D., F.R.C.P.

Another of the few remaining links with the older generation of Edinburgh practitioners has been broken by the passing of Dr. William Taylor. Till within a few days of his death, which took place on 31st July at his residence, 12 Melville Street, his burly figure was well known on our streets, and although he had reached his eightieth year his step, if somewhat shorter, had lost little of its elasticity.

A native of Forfarshire, he spent some years in Australia before he entered on the study of medicine at Edinburgh University, where he graduated M.D. in 1867. With Professor Laycock, then in the chair of the Practice of Medicine, he formed a fast friendship, and for two years he acted as assistant to the Professor and took an active share in the work of teaching. Later he was associated with Professor Spence in the Surgery Department, and conducted with great acceptance the tutorial classes on minor surgery and the practice of bandaging, in which he was exceptionally expert. His wide acquaintance with general surgery was evidenced by the fact that he was entrusted with the task of re-editing Spence's well-known text-book of *Systematic Surgery*, which he did with the greatest care. For some years after severing his connection with the University he conducted private classes on Practical Surgery, which were exceedingly popular.

While still an undergraduate he was placed in charge of the Maternity Hospital for a time, and here he developed an interest in obstetrics which was maintained throughout his professional life. To these pages he contributed in 1865 the record of a case in which he maintained the fœtal circulation in a pair of twins after birth by means of artificial heat in water. Dr. Taylor was an active member of the Obstetrical Society, and took a prominent part in its proceedings. He was also a regular attender of the meetings of the Medico-Chirurgical Society for many years, and although he did not often take part in the discussions, when he did it was always to add some pithy original remarks which were more notable for their sound common sense than for their orthodoxy. The writer recalls an occasion in which he intervened in a discussion on the perennial subject of the action of alcohol. The effects of the drug upon rabbits, guinea-pigs, and mice had been freely quoted, but Dr. Taylor was not satisfied that these observations were conclusive, and pleaded for evidence derived from the administration of alcohol to "large carnivorous animals, such as lions and tigers."

Among his other interests were mechanics and electricity, which he pursued partly as a hobby and partly for professional purposes. He was President of the Royal Scottish Society of Arts, and communicated a number of interesting and valuable papers to the *Transactions* of the Society. He was also a member of the famous old Edinburgh club the Monks of St. Giles, where his genial social qualities found full scope.

For many years he rented Swanston Cottage,

> "A bonny bit,
> 'Atween the muckle Pentland's knees,"

hallowed by its association with Stevenson, and there he spent such spare time as a busy practice allowed him, and collected many interesting souvenirs and recollections of his literary favourite.

RECENT ADVANCES IN MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

W. T. RITCHIE, M.D., EDWIN MATTHEW, M.D., J. D. COMRIE, M.D., AND A. GOODALL, M.D.

Treatment of Cardiac Irregularity.

Ryser (*Correspondenz-Blatt für schweizer Aerzte*, Nos. 24, 25, and 26, June 1916) contributes a clinical study of the phenomena attending irregular activity of the heart in the various forms of this condition. His most interesting deductions are in the matters of prognosis and treatment. With regard to the occasional and isolated extrasystoles which are shown by numerous hearts, sometimes with the consciousness of the subject, sometimes without it, he considers these are of a completely harmless nature. For instance, he considers that these occasional extrasystoles in an organically and otherwise functionally sound heart are no disqualification whatever for military service. As regards treatment of this condition he sets in the first place of importance strychnine, which has also the recommendation of Wenkebach. The dose he gives is 2 mgrs. daily over 20 days. Its effect is usually shown quickly, and lasts for a prolonged period. In those cases which do not respond to strychnine he recommends small doses of digitalis, which diminish the excitability of the heart muscle. He recommends 0·05 to 0·1 grm. of digitalis leaf. Physostigmin he also recommends in minute subcutaneous doses, and in occasional cases quinine may be tried, with advantage. Heart-block is of course a much more serious condition, particularly when it is due to an organic cause, which then probably affects wide areas of the heart as well as the conducting bundle; digitalis has in these cases a positively hurtful action, but atropin, which increases the conductive power, may be beneficial. Auricular flutter accompanied by perpetual arrhythmia is, on the contrary, a condition in which digitalis must be pushed to an extreme, even to the extent of bringing on sickness and great slowing of the heart.

Report on the Enteric Cases at Heliopolis.

Summons (*Med. Journ. of Australia*, 6th May 1916) gives a review of the enteric cases treated in the first Australian General Hospital

at Heliopolis. Few soldiers will have sustained any permanent damage from this infection, and out of 800 cases treated in that hospital there were only 5 deaths. But although the paratyphoid fever was of little harmfulness to the individual it was a great burden to the army and to the medical units. The European and Egyptian predominating organism appeared to be *bacillus paratyphosus* B, while in the Gallipoli infection the predominating organism was *bacillus paratyphosus* A. It is suggested that as *bacillus paratyphosus* A is the common organism in India it may have been brought to Gallipoli by the Indian troops. The writer considers that the value of prophylactic inoculation has been fully upheld, and that the troops having now been inoculated against paratyphoid infections, and carriers having been discovered and isolated there will probably be far fewer cases in future.

Perforating Carcinoma of Œsophagus.

In about 50 per cent. of cases of carcinoma in this region there is perforation into the mediastinum, pleura, bronchi or blood-vessels causing death, but Guttmann and Held (*Med. Record*, 10th June 1916) record a case in which the symptoms of a broncho-œsophageal fistula due to a carcinoma persisted for four months before a fatal result supervened. There was no vomiting, fœtor, dysphagia, or similar symptoms up to within a short period before death, but there were cough, spit, etc., which were afterwards found to be due to a bronchiectasis which clinically had simulated tuberculosis. Coughing and choking supervened whenever the patient attempted to drink, and the dribbling of food down through the fistula into the bronchi could be quite well seen by the fluoroscope during life.

Myopathy and Internal Secretions.

M'Couch and Ludlum give a review of what is known as to the relations subsisting between myopathy and other muscular disturbances on the one hand, and disorders of the internal secretions on the other hand (*ibid.*). They find that myotonia congenita and myasthenia gravis are frequently associated with muscular dystrophy, and that myotonia congenita has occurred in connection with muscular dystrophy and may be identical with one form of it. There is some evidence suggesting that thyroparathyroid deficiency may bear a causal relationship to myotonia, while myasthenia is frequently associated with hyperthyroidism, hypoadrenalism, and probably with hyperactivity of the thymus. Myopathy has been found in association with many disorders of internal secretion, perhaps most frequently with hypopituitarism. The writers report a case which presented the fully developed characters of both dystrophia adiposo-genitalis and the facio-

scapulo-humeral type of progressive muscular dystrophy. Whether the glandular disorders bear a causal relationship to myopathy or are secondary to it, or whether both are due to a common cause, is uncertain, but the writers conclude that the combination is too frequent to be a mere coincidence.

Control of the Pneumothorax Treatment of Pulmonary Tuberculosis.

This treatment has fallen into desuetude largely because of the difficulty of controlling and observing the effect of air introduced into the pleural cavity. Hirsch (*ibid.*) describes how the injection of air should not only be controlled at the time, but should also be carefully and persistently followed up either by repeated radiography or by frequent examination of the lung by means of the fluorescent screen. As regards the subsequent study for the determination of the amount of collapse, restitution, etc., the X ray examination gives the following data. The degree of pneumothorax and its distribution, the condition of the lung as to collapse, mobility, etc.; the condition of the pleural cavity as to the presence of effusion; the displacement, if any, of the heart and other mediastinal contents; the position and movement of the diaphragm; the occurrence of subcutaneous emphysema; the extent to which absorption has taken place, a moderate amount of gas, say 600 c.c., being all absorbed in two weeks; and finally the progress of the disease. J. D. C.

SURGERY.

UNDER THE CHARGE OF

J. W. STRUTHERS, F.R.C.S., and D. P. D. WILKIE, F.R.C.S.

Resection of Joints in War Surgery.

Delagenière (*Journ. de Méd.*, 10th April 1916) describes his experience in a large number of cases of joint injuries and infections which were treated by excision.

In the case of war injuries resection of joints is most often indicated in order to suppress the local infections of the joint, which in many cases may be responsible also for a general septicæmia. In other cases the object may be simply to remove or repair crushed articular surfaces, or in the later stages to correct malposition.

As a general rule, ankylosis is desirable in the case of the knee-, ankle-, and the shoulder-joint; a movable joint is aimed at in the finger-, wrist-, elbow-, and hip-joints, and sometimes in the shoulder.

Hip-Joint.—Resection of this joint is reserved for serious cases where it is necessary to operate in order to save life endangered by septic absorption. Before proceeding to resect for infection of the hip-joint it is advisable to try the effects of drainage both from the anterior and posterior aspects of the joint. When these measures fail it is necessary to resect.

This operation was performed in six cases, and the author not only saved all his patients, but obtained better functional results than he had expected. A posterior incision was used, reaching from the posterior superior iliac spine to the great trochanter, the attachment of muscles to the latter being carefully preserved. The neck of the femur is best divided close to the trochanter, after dislocating the head of the bone backwards; the acetabulum is now exposed, and diseased or loose fragments of bone can be removed. If the tissues are offensive, pure carbolic acid is applied. The wound is partly closed after ample provision for posterior drainage.

The subsequent attitude of the limb is important: the upper end of the trochanter with its muscles should be pushed well into the acetabulum, and the limb then fixed in pronounced abduction by a plaster casing which should extend from pelvis to foot, a window being left for the changing of dressings. In five of the six cases thus operated on a moderate amount of movement was present, and the patients were able subsequently to walk comfortably, with slight abduction of the limb and with the foot everted.

Knee-Joint.—To obtain a good result in the knee, ankylosis must be osseous and the limb should be straight without much loss of substance. If too much bone has to be sacrificed on account of the disease or damage, the result may be bad. In order to maintain greater length it may be possible to use for the ankylosis one condyle alone, if the other is destroyed; in such cases special care has to be taken afterwards to prevent displacements. Excision of the knee-joint was performed in twenty-one cases. These were all cases of suppurating joints which had been drained by other surgeons without success and without being sufficiently immobilised; in most of the cases also there was a degree of septicæmia. In such cases amputation of the limb or excision of the joint are the only alternatives, and the author considers that excision is the less taxing of the two.

In recent cases it is advisable to examine the joint with the X-rays in the first instance, and then, if the infection is not severe, to drain the joint low down on either side; afterwards the limb should be immobilised. When the bony lesions are marked, or if the infection is already severe, excision of the joint should be done at once. In the case of foreign bodies the procedure recommended is to open freely by a transverse incision across the patellar tendon and to leave the joint open; later, secondary suture can be done or the joint may be excised.

The author employs a horseshoe-shaped incision for excision of the knee joint. The patella is removed, and the ends of the bone are sawn across just beyond the diseased or damaged part; silver wire is used for suturing the bones together. Pockets of pus should be carefully looked for in the supra-patellar pouch and towards the back of the joint, and separately drained.

In infected cases the wound is left open, or a single stitch may be placed to unite the cut edges at the centre of the flap. In spite of the presence of pus, union is rarely delayed beyond two months, although retraction of the flap may prevent early healing. In a series of twenty-one cases all the patients survived with a useful limb, with the exception of one patient, who died six months later from the original septicæmia.

Shoulder-Joint.—The indications for excision are necrosis or comminution of the head of the humerus, and ankylosis associated with necrosis and fistulæ. The author has tried both anterior and posterior routes of access, but, unless enlargement of the original wound is sufficient, prefers the anterior incision. When the articular surface alone requires removal the results are excellent, but if the bone has to be divided below the surgical neck the results are bad. In cases of ankylosis, just sufficient bone should be removed to give free movement, and exercise must be commenced soon after the operation. Excision of the shoulder-joint was performed in fourteen patients, all of whom survived with a varying degree of movement, although the author does not differentiate between movements at the shoulder-joint and of the scapula.

Elbow-Joint.—The main difficulty in this joint is to know how much bone to remove, as, especially in young soldiers, there is a great tendency for new bone formation and ankylosis. The best level to divide the humerus, according to the author, is just below the condyles; the olecranon should always be removed, preferably on a level with the articular surface of the radius. The author prefers a posterior incision, and in the case of infected joints the wound should be left open. Seventeen patients were operated on, and all survived; in most of the cases, however, the subsequent movements at the elbow-joint were limited.

Wrist-Joint.—The author has had experience of six cases. A posterior incision was used, and one or both rows of carpal bones were excised. Excellent results as regards movement were obtained in cases of recent infection, when exercises were begun soon after the operation. When the tendons and wrist-joint were stiff, from the effects of an old-standing infection, the results of excision as regards movement were disappointing. In a chronic case of this kind a more favourable result was achieved by the grafting of a portion of fat-bearing tissue between the ends of the bones.

Saphено-Peritoneal Anastomosis for Ascites.

Ruotte in 1907 suggested that ascitic fluid might be drained off into the general circulation by means of a union between the saphenous vein and the peritoneal cavity. It was proposed also to do the operation on both sides at one or two sittings. About twenty of these cases have been reported, chiefly from continental clinics. No attempt in this series was made to select the cases, and as the operators were not specially skilled in blood-vessel suture the results must be considered distinctly encouraging, a marked improvement following in one half of the cases. In some cases patients who had been bedridden were able to resume their occupations.

The type of case suitable for this operation is the ascitic patient, with portal obstruction, and who is unable to stand the constant loss of fluid necessitated by repeated tappings. As compared with the Talma-Morrison operation, the operation of saphenoperitoneal anastomosis is much less severe, and can be done under local anæsthesia.

Bernheim (*Amer. Journ. Med. Sci.*, June 1916) records a case which is interesting on account of the technique adopted, although, unfortunately, the patient died one week after the operation. In this particular case a Talma-Morrison operation had been previously tried unsuccessfully. The operation was rendered easier as the sac of an inguinal hernia was present on the left side.

Under local anæsthesia 6 inches of the saphena vein were exposed, and at the lower end of the wound the vein was divided and both ends tied. The peritoneal sac was next exposed by a 2 inch incision placed over the external abdominal ring. A small hole was made in the sac, and two gallons of fluid were drained off. The next step was to undermine the skin between the two incisions, and to pull upwards the lower end of the vein beneath the skin so that it lay close beside the peritoneal sac. A lateral anastomosis was then completed between the vein and the peritoneal opening, a fine straight needle and blood-vessel silk being employed. Prior to the suture it was noted that the valves were competent, as there was no bleeding. At the same time the patency of the vein was demonstrated by injecting upwards a small amount of saline solution. A lateral anastomosis was preferred, as the junction could thus be more easily made, and also the opening could be made larger than with an end-to-end union.

The operation was confined to one side, as the other leg was œdematous. The patient did well after the operation, in spite of a slight leakage of ascitic fluid, but died about a week later, with symptoms suggestive of morphia poisoning. There was nothing in the clinical features to suggest that the operation had been the cause of death.

A Review of Over Fourteen Thousand Surgical Anesthesias.

Alice Magaw (*Western Medical News*, May 1916), anæsthetist to the Mayo Clinic, gives her experience in general anæsthesia. At the St. Mary's Hospital in Rochester preference has always been given to open ether. In 1905, out of 3080 anæsthesias ether was administered in 2847. In 14,380 anæsthesias the author has had no death directly due to the anæsthetic. The preference for ether as the anæsthetic has become greater every year, and the open method of administration is the method of choice. Other methods of inducing, or as preliminaries to, general anæsthesia have been thoroughly tried, such as nitrous oxide gas or injection of scopolamine and morphia, but these modifications have proved less satisfactory on the whole than the drop method, which has now been used for ten years. In goitre cases $\frac{1}{4}$th gr. of morphia and $\frac{1}{150}$th gr. of atropin are given half an hour before the operation.

The author employs two 4-ounce ether cans, one with a large dropper to be used till the patient is under, and the other with a small dropper to be employed during the course of the operation. An ordinary cork is fitted into the can with a groove cut on either side, one groove being filled with absorbent cotton-wool, which should project for an inch beyond the cork.

An improved Esmarch's inhaler is used with two thicknesses of stockinet. The ether is first administered slowly in drops, as in chloroform anæsthesia, until the face is flushed, and then a few layers of gauze are added and the ether given a trifle faster until the patient is under; thereafter return is made to the same covering as at first, and a regular drop is continued during the operation. A much deeper narcosis is needed to start an operation than during its progress. Only the inexperienced take the pulse or touch the cornea during an operation when giving ether.

Suggestion is a great aid in producing a comfortable narcosis. The anæsthetist must inspire confidence and study the temperament in each case, deciding which mode of suggestion will be most efficacious—the abrupt, crude, and very firm, or the reasonable, sensible, and natural. In most cases the latter mode is by far the best. The patient should be "talked to sleep" with the addition of as little ether as possible, but the patient himself should not be allowed to talk, as by so doing he is more apt to become boisterous; further, the patient should never be told to breathe deeply, as this induces a feeling of suffocation and a tendency to struggling. Patients can be brought under ether in this way in from three to five minutes, and they do better if the operation is started at once. There is no single infallible sign of complete anæsthesia. If the jaw is relaxed and in place, respiration deep and regular, colour normal, quality of pulse good, then there need be no fear about the rate of the pulse or the pupils.

During thirteen years at St. Mary's Hospital all patients have been anæsthetised on the table in the operating-room, and preparation of the patient has been carried out at the same time. The simultaneous preparation of the patient is one of the important factors in inducing anæsthesia, for it diverts the patient's attention and less anæsthetic is needed.

The position of the patient should be adjusted during the preparation. It is an advantage, for instance, to place the patient in the Trendelenburg position before narcosis is completed, as the deep respiration helps to empty the pelvis, and the intestines can be more easily packed off when the abdomen is opened. J. M. G.

OBSTETRICS AND GYNECOLOGY.

UNDER THE CHARGE OF

A. H. F. BARBOUR, M.D., AND J. W. BALLANTYNE, M.D.

Clinical Significance of Lutein Cysts of the Ovaries.

Professor Paul Bar has an article (*Arch. mens. d'obstét. et de gynec.*, ann. v. pp. 49-58, January-March 1916) on the clinical significance of lutein cysts of the ovaries developing after the removal of a hydatidiform mole of the uterus, and Professor Bar never writes on any subject connected with either the physiology or the pathology of pregnancy without aiding in the elucidation of some of the many problems with which these subjects teem. It is so in the present instance, although as a matter of fact the particular problem dealt with is one of the most obscure. There is, indeed, so much that is obscure about hydatidiform moles, about lutein cysts of the ovaries, and about their association together, as to justify the use of the word mysterious: and if one adds to these the problem of the chorio-epithelioma and the origin of malignancy the mystery becomes very dark. The clearing up of the mystery will yet be accomplished, and in the meantime every advance in that direction deserves to be chronicled.

Is there more than an accidental relationship between the mole and the ovarian luteinic cysts? There are not yet great numbers of published cases, but Bar thinks that the fact that 59 per cent. of lutein cysts accompany hydatidiform moles negatives the idea of fortuitous coincidence, and it is difficult to avoid such a conclusion. But what is the nature of the bond or connecting link? Professor Bar turns first to physiology and states the aphorism (it may almost be called):

It is not doubtful that the harmonious development of the villous and decidual elements which characterises the regular implantation of a normal ovum is in relation with the modifications in the ovisac which occur in pregnancy, whether it dominates them or they dominate it. When a mole develops, whether the villous change be primary or caused by an abnormal decidual reaction, the cellular foetal elements are both altered in their relations to other structures and in themselves; everything in the implantation is thrown out of order, and in such cases it is justifiable to institute a comparison between villous elements embedding themselves in the maternal parts and cancerous elements grafting themselves upon an organism and developing there. Therefore one may go further and expect to find ovarian morbid changes associated with so pathological a state of affairs. Consequently Bar says: "It experience prove that the appearance of lutein cysts in the ovaries and their rapid development in cases of hydatidiform mole are not accidental facts, may one not hope to find by their investigation the demonstration (by pathology) of the intimate relations which exist between the ovarian functions, those of the uterine mucous membrane, and the normal implantation of the ovum?" The whole problem, however, is not dealt with here, Professor Bar preferring to give details of a case and to draw some important clinical deductions.

Bar's case was similar to that recently presented by Couvelaire to the Obstetrical Society of Paris. The patient, a young woman, had a hydatidiform mole removed from her uterus; the removal had apparently been complete, and it had been noted by the surgeons at the time that the uterine annexa were not the seat of palpable lesions. Hemorrhage from the uterus continued, and then, on a new examination, the surgeons were surprised to find papillary growths behind and at the sides of the uterus, which remained enlarged. The patient came to Ricard and Bar, who diagnosed ovarian cysts following upon a hydatid mole pregnancy. Ricard removed the cysts and the uterus, and found inside the latter and attached to its posterior wall a small mass like a placental polypus; the ovaries were transformed into masses of cysts, with thin walls and clear contents, arranged like bunches of grapes. Full details of the histological changes need not be given, but there was a very great hyperplasia of the lutein elements. The little polypus showed hydatid vesicles which burrowed deeply into the uterine tissue and were covered by the cells of Langhans' layer and by syncytium, both immensely overgrown. It is possible that the lutein cysts may have been present when the mole was removed, although, if so, they must then have been extremely small; but it is at any rate certain that they grew with swift rapidity after its evacuation and whilst the molar villi were still attached to and growing in the uterine wall. Couvelaire's case was exactly similar. Other authors have reported the complementary phenomenon, viz. the

reduction in size, almost to disappearance, of co-existing lutein cysts after the removal of a hydatidiform mole. Professor Bar concludes that the moles most likely to be associated with changes in the ovaries are those which develop very early, are generalised, have no embryo in them, and show a thoroughly disordered state of their epithelium.

The clinical conclusions are of great importance. They are these: (1) When, associated with a mole, the presence of ovarian cysts is recognised, it is prudent not to be content with the removal of the mole; indeed the operation of hysterectomy seems to be indicated, for the possibility of a burrowing or invading mole is particularly to be feared. (2) When, after the removal of a mole, ovarian cysts continue to develop, or, still more, when they make their appearance, one ought to dread the growth of molar elements in the uterine wall. (3) The significance of ovarian cysts, just referred to, becomes still more pressing when the uterine hæmorrhages persist, demanding rapid operation even when other circumstances are against it, and necessitating hysterectomy. Certainly in view of such cases as Bar's it will be unwise on the part of an obstetrician not to examine carefully for signs of ovarian tumours in every case of hydatidiform mole.

Nitrogen Metabolism during Pregnancy.

Dr. Karl M. Wilson (*Bull. Johns Hopkins Hosp.*, xxvii. pp. 121-129, May 1916) rightly deplores the lack of observations on metabolism in early pregnancies; but indeed the investigation of the chemistry of pregnancy, both late and early, has been long delayed. After touching upon experiments made on pregnant animals, he refers to observations on pregnant women (all in the second half of gestation) carried out by Schrader, Slemons, Hahl, Bar, and a few others, and he quotes Hahl's interesting conclusion: "Normal pregnancy in a healthy maternal organism, living under normal exterior conditions, produces a marked retention of nutritive materials, which suffices for the development of the product of conception and its annexa, for the modification of the generative organs and the formation of a reserve fund which will be utilised during labour and the puerperium; normal pregnancy does not then constitute for the mother a period of loss, nor yet is it for her a period of gain." Dr. Wilson was able to apply himself to the careful observation of three pregnant patients, one from the tenth to the fourteenth week, one for the last 133 days, and one for the last 101 days of gestation. Details are given as to the food supplied to these women, their exercise, the analysis of the urine and fæces, the weighing, etc., and these must be referred to by workers along similar lines and engaged in similar investigations; but it will suffice for present purposes that Dr. Wilson's results be stated as conclusions (without figures and minutiæ). His first conclusion is

that in the perfectly normal pregnant woman storage of nitrogen begins at a much earlier period than has hitherto been supposed; possibly the organism may acquire the capacity for storing nitrogen from the very beginning of the pregnancy. In the early months this storage is far in excess of the actual needs of the developing ovum, and the excess must be added to the general maternal organism. Storage of nitrogen continues throughout the entire duration of pregnancy, being most marked during the last few weeks, when the total needs are at a maximum. The nitrogen stored is greatly in excess of the needs of the developing ovum, so that, apart from the amount needed for the hypertrophy and development of the genitalia and breasts, a large proportion of the nitrogen stored is added to the general maternal organism as "Rest material," though, concerning the form in which this reserve is stored, Dr. Wilson is unable to make any positive statement. The nitrogen capital of the maternal organism is thus increased, though the reserve supply may possibly be entirely exhausted during the puerperium and period of lactation. Then follows the big deduction (which resembles Bar's rather than Hahl's): "In the healthy woman who goes through a normal pregnancy the period of gestation does not necessarily represent a sacrifice of the individual for the sake of the species," but may actually be a period of gain. There is a relative increase in the percentage of urinary nitrogen excreted in the form of free amino acids, though not necessarily an absolute increase in this form of nitrogen. The last conclusion is that there is also a tendency for the percentage of ammonia nitrogen to become increased during the last weeks of pregnancy, although at other times during the pregnancy there is practically no variation from the percentages noted in non-pregnant individuals upon a similar diet. As stated above, Dr. Wilson was unable definitely to say what form the stored "Rest material" takes; but he suggests that a certain portion is made use of in the formation of new blood, the blood volume being markedly increased at the end of pregnancy; another portion is probably utilised in the hypertrophy of muscles and the various glandular structures affected by gestation; and possibly the remainder is placed in the form of unorganised protein as a reserve, probably to be drawn upon extensively during the puerperium and lactation times. Another interesting matter discussed by Wilson was the effect which recurring menstrual days may have on metabolism during pregnancy; he thought that cyclical variations in the nitrogen exchange might then occur and find thus their explanation, but he was forced to abandon this view, so far at any rate as the three patients examined were concerned. By a simple series of calculations the nitrogen content of the ovum was arrived at. In one of the patients it worked out as follows: Child, 62·10 gm. N.; placenta, 14·66; cord and membranes, 2·00; and amniotic fluid, 0·19 gm. The total nitrogen

content thus was 78·95 gm. This was in the patient who was under observation for the last 133 days of pregnancy. The balance added to the general maternal organism was 345·84 gm. J. W. B.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

W. G. SYM, F.R.C.S., AND A. MacGILLIVRAY, M.D., D.Sc.

The Refraction of Myopes—A Consideration of Current American Teachings.

In the hope of clearing up to some extent the confusion which beginners in refraction work experience from the variety of opinions they hear in regard to myopia, Dr. Emory Hill, Chicago (*The Ophthalmic Record*, May 1916), sent a list of questions to 130 teachers of ophthalmology in American schools, from whose answers certain interesting tabulations are recorded. The questions were necessarily of a general sort, to which categorical answers could not be given. Some questions could be answered only after prolonged search through case records covering a period of years. Seventy-eight replies were received, varying from brief statements of general rules followed in refraction of myopia and myopic astigmatism to elaborate answers in the form of letters which were of such value as to warrant extensive quotation.

With regard to the use of cycloplegia, the important deduction, warranted from the answers received, is that American ophthalmologists believe in cycloplegia. Thus 73 of 78 men use some cycloplegia in myopic children, 52 of these use a stronger drug than homatropin, while 44 prefer to use atropin, the strongest of cycloplegics. In the case of adults less than 30 years of age, 70 of the 78 men use some cycloplegic, 16 use a stronger drug than homatropin, while 11 prefer atropin. In the group of adults between 30 and 40 years of age some cycloplegic is insisted upon by 60 of the 78 men, something stronger than homatropin is used by 6, while atropin is preferred by 5. In adults between 40 and 50 years of age, 39 (one-half of the entire number) use some cycloplegia, homatropin being the favourite drug in this and the preceding group. Even in patients beyond the age of 50, 17 men use a cycloplegic.

With regard to making a full correction of astigmatism associated with myopia, it was noted that 50 gave the full astigmatic correction. Twenty-three did the same except in the case of very high astigmatism, where they gave a partial correction. Two usually cut the astigmatic correction slightly, and 3 habitually gave only a partial correction for

astigmatism. Thus 73 of the 78 replies indicated the desirability of correcting as fully as possible the existing astigmatism, a minority granting the desire of the patient to have the glass which gave more immediate comfort when the astigmatic error was high. This applied generally to individuals who have not worn glasses in youth.

With regard to the correction of myopia according to the amount of error, the tables submitted are very instructive. The majority of 78 ophthalmologists are in the habit of giving the full myopic correction except in the rare cases of very high errors. A considerable number make only a trifling reduction from the full correction, and two men habitually over-correct myopes. To this showing in favour of a practically full correction must be added the observation that the heading, "All that age and accommodation will allow with comfort," generally means (as a reading of the full replies would show) as close an approximation to the full correction as is possible in each case. The men who answered in this way often supplemented their answers by emphasising the ideal of full correction which they could not attain in cases which were neglected in youth. Scarcely 10 per cent. of the replies indicate a disregard of the principle of full correction.

With regard to the query as to every myope wearing glasses for distance and near, the following is a summary:—More than 50 per cent. of the 78 men consider that all myopes should wear glasses for distance, while nearly 50 per cent. think that all myopes should wear glasses for near. The diversity of opinion on these questions is less than the table would indicate, if one considers the exceptions made to the general rule of prescribing lenses for these cases. For example, patients with very small errors (0·25 or 0·50); patients with no astigmatic error; patients who report every few months for observation; patients who object strongly to wearing glasses on the street; patients whose myopia is precisely balanced by their presbyopia, etc. After allowing these exceptions there remains the great body of myopic individuals for whom the ophthalmologists quoted would prescribe glasses for constant use, if the interpretation of their replies be correct.

The proportion of cases wearing one correction for all purposes is very instructive, as the following summary will show:—Fifty-seven men did not answer separately, since their reply to the previous question indicated that ability to wear one pair of glasses for all purposes was largely a matter of youth. Some emphasised the factor of small errors and previous adequate correction. It seems to be agreed that individuals with high errors, and those who have had no glasses or very partial corrections in youth, have difficulty in maintaining accommodation for near vision with an adequate far-seeing glass. Twenty-one replies to this question specified that the young, and the cases of low error, wear one pair of glasses for all purposes.

With regard to the method of preventing an increase of myopia, the majority of replies to this question indicate satisfaction on the part of the ophthalmologist with his method, while a few express no enthusiasm for any method, and feel that the problem of myopia has not been solved, and even that its solution is divorced from the realm of refraction. Forty-one men believe that the use of cycloplegia extensively and the constant use of the full myopic and astigmatic correction are the surest preventives of increase of myopia. Four men express faith in the opposite method of scant use of cycloplegia and wearing only a partial correction. Two men do not use cycloplegia, but prescribe full corrections. One man emphasises the value of giving a restful near glass, and always prescribes bifocals. This he believes to be the secret of success. Five men emphasise the importance of a full correction of astigmatism and of hyperphoria, together with attention to hygiene. One man says that all myopes must give up near work. Two men feel that when myopia develops in youth it will increase in spite of all treatment. Two men state that they do not see progressive myopia. One man has seen only one case of "malignant" myopia in 15 years. Three men think that glasses have no influence upon myopia. Three men regard heredity as the principal factor. Thirteen attempt no answer to the question.

It is unsafe to draw hard and fast conclusions from a small number of observations, especially when the personal bias of the one reviewing the data enters into judgment. Allowing for these factors, Dr. Emory Hill believes that the following statements may fairly be made as a resumé of the teachings of the 78 ophthalmologists whose views have been tabulated :—

American teachers of ophthalmology believe in the thorough use of cycloplegia in the refraction of myopes (more than 90 per cent.).

They believe in the full correction of the astigmatism existing in myopic cases (more than 90 per cent.). The exceptions to this rule are in the very high errors which have not been corrected in youth.

A large majority regard the full correction of myopia as the ideal to be sought, while many think this ideal unattainable in cases which have gone through childhood and early adult life with no glasses or inadequate glasses.

A majority think that myopes should wear glasses constantly. A majority expect patients under the presbyopic age to wear one correction for all purposes. A majority regard the wearing of the full myopic and astigmatic correction, based upon the findings under complete cycloplegia, to be the surest means of preventing the progressive increase of myopia.

Among the men whose opinions are highly valued in the American Academy of Ophthalmology and Oto Laryngology some have written their views at length. The following remarks were selected from the

replies of Drs. Alexander Duane, John Green, jun., Edward Jackson, Samuel D. Risley, Samuel Theobald, Casey Wood, and Hiram Woods:—

"I consider it exceedingly important that the ciliary muscle be completely under the influence of the cycloplegic, and the choroidal congestion be allowed to subside before refraction is attempted. Numerous cases come into my hands in which myopia has been overcorrected by opticians and, I regret to say, by oculists who use cycloplegia carelessly or not at all."

The correction "depends on visual acuity, condition of fundus, age, and finally determining how much the patient will take comfortably. I come as near total correction as possible, if the fundus is good and if accommodation develops. If it does not, I reduce the correction to the point of comfortable tolerance."

"I have regarded myopia as pathological . . . it must be treated from that point of view; in correcting the refraction error a cycloplegic is just as necessary in myopia as in hypermetropia; that since the myopic eye is a pathologic eye the cycloplegics are even more necessary because of their remedial influence over the pathologic conditions of the uveal tract. I have never hesitated to use the atropia or hyoscyamin sulphate three or four times daily for long periods of time at any age or any degree of myopia or myopic astigmatism in more or less malignant choroidal conditions, but have often employed at the same time weak solutions of eserin salicylate (gr. $\frac{1}{20}$ to $\frac{1}{10}$ to fluid ounce 1) twice daily, not to counteract the mydriasis of the cycloplegic, but because of the physiologic influence in contracting the dilated anterior ciliary vessels. . . . I prescribe full corrections of the existing astigmatism. Many myopes have high degree of hyperphoria. This I have always been careful to correct either with vertical prisms or by cautious tenotomies. . . . After 50 years of age I prefer homatropin solutions three or four times daily and the eserin solution at bedtime and in the morning."

"In practically all my refraction cases, from 8 to 45 and in nearly all examined for the first time between 45 and 49, I use homatropin. I make no distinction in this regard between myopia and hypermetropia. In cases over 40, and in others too, if I think there is any possibility of glaucoma, I test the tension before recommending homatropin. . . . In cases in which the range of accommodation remains high (above 10) in spite of repeated instillations of homatropin, or in which the results are usually varying, or in which skiascopy shows a varying, evidently unrelaxed, accommodation, I proceed to the use of atropin just as for children under 8. And I find it necessary to do this fully as often in the case of persons over 40 as in younger people. It has been my experience that it is just in these older subjects that it is hard to get the accommodation to relax. . . . My cardinal principle in applying the correction in myopia is thus stated: Whenever possible the patient

should wear (*a*) the full correction of his myopia and (*b*) wear the same correction for distance and near. . . . My experience has been that in myopia of under 10D I can in almost every case, and in myopia of 10-15D I can in a vast majority of cases, prescribe the full correction with satisfaction, comfort, and advantage to the patient. This is occasionally possible in patients over 15D. . . . Of course, the younger the patient the easier it is to apply the full correction. But in older subjects seen for the first time I should always feel inclined to order the full correction experimentally, believing that in the great majority of cases the experiment would succeed, and that, if successful, it would afford the patient more relief than would an undercorrection."

"Moderation in the near use of the eyes, proper lighting, and avoidance of the stooping position, are important; but far more important is the correction of accompanying astigmatism, anisometropia, and heterophoria, both in far and near, but especially in near vision."

"I am sure that, since I have treated young myopes with total correction, provided they have good fundi and normal vision, I have seen fewer cases of progressive myopia. . . . I do not see how one can come anywhere near accuracy without cycloplegics."

"Under the continuous wearing of the full correction increase of myopia is rare at any age and under any circumstances when it occurs in cases after repeated correction."

A different emphasis on treatment is indicated in the next quotation: "In treating myopia of the (possible) progressive variety the use of glasses is but one—and a relatively small—factor. To be rigidly insisted upon in addition are the usual rules of ocular hygiene. In the hereditary type especially, care in the amount and time of reading and other near work, outdoor life, illumination, the state of the health, use of atropia, tinted lenses, oculomotor exercise, sort of type of books, etc., are quite as important. In about one-third of these cases in particular I am unable (for various reasons that might well fill a volume) to prevent the slow but certain progress of the myopia. I console myself with thinking, however, that if nothing had been done the 4D or 5D myope might have gone on to 10D or more."

In conclusion, Dr. Hill emphasises that a majority of 78 American teachers of ophthalmology believe in a painstaking and time-consuming scientific method of refracting myopes, and moreover, that this method is advocated with enthusiasm by a considerable group of those whose positions as leaders in our specialty entitle their views to the utmost respect. His brief investigation reveals no extensive oppositions to these views, yet here and there conflicting teachings are producing confusion in the minds of students and bringing discredit upon the profession, who should exhibit some uniformity in a practice which is supposed to be based upon an exact science. A. MacG.

THERAPEUTICS.

UNDER THE CHARGE OF

JOHN EASON, M.D.

Treatment of Pneumonia.

Solis Cohen in a recent paper states the principles of what he names the *rational* treatment of pneumonia. Neutralisation, antagonisation, counteraction, elimination, and specific stimulation have to be attempted. Stated more fully these are :—

1. Neutralisation of the toxic agents present.

2. So far as neutralisation fails, antagonisation of the toxic actions of those agents.

3. In so far as antagonisation fails, counteraction of the toxic effects.

4. Free elimination, to carry off as rapidly as possible the compounds, probably incompletely neutralised, formed by toxic and antitoxic agents; together with the effete products of normal and abnormal metabolism and tissue change.

5. Specific stimulation of defensive and restorative tissue changes. In addition, certain other measures are suggested by considerations of a somewhat empirical nature.

6. The theory of F. P. Henry, that a decreased elimination of urinary chlorides indicates that the natural defence is utilising these compounds, and his expedient of saline infusion to supply additional chlorides; the traditional use of ammonium chloride; and Quimby's good results with the administration of a mixture of ammonium, sodium, and calcium chlorides by mouth, are at least suggestive.

7. Observation in many forms of infectious disease has convinced Cohen that patients do better not only when they are excreting large quantities of urine, but when the urine is alkaline. In other words, there is in most an acidæmia; and a crude index to its neutralisation is afforded by the urinary reaction. Hence alkaline salts are associated with the chlorides, whether given by the mouth or by rectal or subcutaneous infusion.

8. Finally, it would be highly desirable to have also a bacterial agent, general or specific. Bacteriology or chemistry may yet give us this. Optochin has its advocates. Iodine in its colloidal state may possibly have some potency. Creosote and phenol compounds may have some value. It is not to be forgotten that the pneumonia poison is probably an endotoxin, set free by the death of the cocci. One may, therefore, do harm by inducing an excessive bacteriolysis.

1. *The Antitoxic Agent.*—While awaiting the antitoxic serum or immunising bacterin we may make use of quinine or of digitalis,

possibly also of veratrum viride. Cohen believes we have already convincing clinical evidence as to quinine: very suggestive clinical evidence as to digitalis: and clinical evidence provoking inquiry as to veratrum viride. Clinicians who have used quinine agree in the observation that in practically all cases, not excepting those of fatal issue, the clinical picture is completely changed. This is especially marked in the freedom from cough and from pain, so that neither expectorants nor opiates have to be used: in the relative ease of respiration, even when the rate is but little lessened, so that oxygen is but rarely required: in the comparative rarity of insomnia, and the frequent relief of delirium. In these cases also termination by crisis is rare. Lysis takes its place. Cinchonism does not occur. In addition to these negative points there are observable positive effects. In many the temperature promptly falls after a single injection of quinine—1·0 to 1·5 gram of the quinine and urea salt in 25 to 50 per cent. solution in hot water—and does not rise again, or at least exhibit a sustained rise. With this, the pulse, the respiration, and unfortunately the blood-pressure, also fall. This is precisely as in the natural and phylacogen crisis.

The tendency to depress the blood-pressure is one reason for avoiding excessive doses or too prolonged use of quinine. Close observation and good judgment are required. Sometimes a single dose brings the temperature permanently below 102·2° F., whilst in others repeated doses of quinine reduce it but slightly. Cohen's rule at present is to repeat the injection—or to give the drug by the mouth—but with smaller and smaller doses (0·5 to 0·1 gram) every third hour until the temperature falls to 102·2° F.; and to repeat the dose—other things being equal—whenever there is a renewed rise to 103° F. This may need to be kept up for two or three days or longer; but often for less than twenty-four hours. If three or four doses have been given in succession without persistent effect on the temperature, the intervals are lengthened, commonly to six hours. The number of injections throughout the whole case varies from one to ten, although in extreme cases Cohen has given fifteen. The average number of doses is four or five spread over twenty-four or forty-eight hours. The temperature of the patient is not the only guide. The general condition of the patient is also to be considered. It is not employed as an antipyretic *per se*, but with the idea of neutralising the poisons that give rise to pyrexia among other toxic phenomena; and the antipyretic effect is taken merely as an index to its general efficacy.

In selecting the temperature, 102·2°, three things were kept in mind. First, the necessity of giving nurses some definite guide for the repetition of doses; second, the statistics of Fenwick, showing that in 1000 cases the patients with a general range of temperature between

102 and 103 F. did better than those with either a much higher or lower range; third, the fact that 104 F. is a temperature lethal to pneumococci *in vitro*. The quinine may be given even when the temperature is below 102·2 F. when the other symptoms so indicate, and especially when the patient is seen first. Again, it is sometimes better to omit it even when the temperature is over 103 F. Indications for the omission of quinine are tinnitus, sweating, visual disturbances, or other signs of cinchonism. As already stated, cinchonism is extremely rare, and Cohen regards this as the strongest evidence of the antitoxic or toxin neutralising effect of the remedy. Quinine is apparently neutralised by something in the pneumonia patient's blood. Cohen believes it may be fairly assumed that the quinine and the pneumonia poison neutralise one another, otherwise the indubitable beneficial action of the quinine urea salt is difficult to explain. In the test tube the drug is highly toxic to many varieties of pneumococcus, but the clinical effect does not seem to be chiefly bactericidal. There is no evident effect on the tissue changes. The physical signs pursue their ordinary course, thus indicating that the morbid and recuperative histological processes are alike unaffected; and cases coming to autopsy exhibit no essential differences from cases in which the death occurs under expectant management, except that with a longer survival they show more extensive lesions. In other words, a greater number of patients with moderate and severe lesions recover. The quinine treatment is not associated with any exemptions from complications. A significant fact is the increased number of cases of delayed resolution. Cohen's very confident assumption is that such cases would have been buried under the expectant management. He says the phenomenon is exactly parallel with the increased number of cases of post-diphtheritic paralysis that followed the early use of antitoxin. Patients badly poisoned recovered in a condition of partial paralysis, instead of being buried in a state of complete paralysis.

When certain optochin, synthetic products, as ethyl-hydrocuprein and hydroquinine (methyl hydrocuprein) become available they may prove to be equal or preferable to the quinine and urea salt. It may well be that the intravenous route will be preferable to the intramuscular. For this the urea compound is not altogether eligible. For the present Cohen uses the carbamide. A correct and sufficient dose may be anywhere from 10 to 250 grs. in the course of the case: the larger quantity being spread over several days.

2. *Antagonists to Toxics.*—The toxic action of the pneumonia poison appears chiefly on the heart; and although the antitoxic agent, quinine, diminishes the danger of heart failure, it does not completely avert it. Cohen's rules for the application of remedies that are not to be prescribed for routine use at fixed intervals are:—First, to inject the pressor agent, commonly cocaine hydrochloride one half grain or more,

or a preparation of the posterior pituitary body with the initial dose of quinine; and second, to repeat the injection every third hour, whenever the systolic pressure curve falls five points below the numeral of the pulse curve. Sometimes the indications may be urgent for more frequent repetition. Sometimes six hours may elapse between successive doses. In some cases cocaine and pituitrine are alternated. If more frequent intervention appears to be necessary, camphor is added. The 20 per cent. solution in sterilised olive oil is used by preference; and of this 2 c.c. or more may be injected, and repeated as necessary—say every three hours; the routine in that case being camphor, one hour, pituitrine the next, cocaine the next, and so on. Camphor might be used alone if the heart rather than the peripheral circulation appeared at fault. Cohen sees no reason to fear large doses of camphor, and understands why some physicians depend chiefly on this drug. Musk is also sometimes used by Cohen. The proper use of drugs to support the circulation is the most difficult part of the treatment. First must be mentioned digitalis. He is afraid of strophanthin.

Digitalis used in small doses in pneumonia is ordinarily without apparent effect. But digitalis used in enormous doses in pneumonia lowers the temperature, reduces the pulse-rate, strengthens the heart-beats, and sometimes raises the blood pressure. This is sometimes more apparent in the diastolic than in the systolic pressure curve. Whenever there is an unduly diastolic pressure digitalis should be given and its characteristic effects on heart, pulse, and respiration will result, provided that quinine has been administered for its anti-toxic action. Cohen believes that ordinary doses of digitalis fail to produce any action on the heart in pneumonia, because such amounts are neutralised by the pneumonia poison. For this reason those who do not use quinine have resort to enormous doses of digitalis. But if quinine is used in advance of digitalis, the neutralisation would appear to have been effected—at least in part—and a smaller quantity of the foxglove preparation seems to produce the result. Cohen prefers to use a mixture of the tincture and infusion, as he does not know which principle, if any, acts as an antitoxic in pneumonia.

3. *Agents of Counteraction.* What is particularly meant by counteraction is the attempt to overcome the ill effects of certain accidents which we have failed to prevent, especially abdominal distension and dilatation of the right heart with consequent pulmonary œdema. In the latter instance blood letting is the best measure. Pulse weakness and low blood-pressure are not contra-indications, but rather emphasise its necessity under the conditions stated. After bleeding it will usually be found that digitalis and camphor have regained their efficiency. Oxygen is also used after venesection.

The oxygen must be used generously for hours.

Dilatation of the stomach is a somewhat rare accident, but perhaps occurs more frequently than is recognised. The use of pituitrin tends to reduce the frequency of tympanites. As soon as the first indication of distension of the abdomen is observed, the pituitrin injections are repeated hourly for at least three hours. Camphor and musk are also of considerable value. From ordinary intestinal distension recovery is frequent, but recovery from dilatation of the stomach is exceptional, unless it is recognised early and treated.

4. *Measures of Elimination.*—Empirically it is known that all febrile patients, and pneumonia patients in particular, do better when the urine is kept alkaline as well as free. Large quantities of water containing an alkaline diuretic, such as sodium or potassium citrate, are therefore given usually in association with sodium phosphate and sodium chloride. Sometimes sodium bicarbonate is used, and citric acid, in the form of lemon juice, added to produce effervescence. The patient is also given as much plain water as he will take. Such treatment is contra-indicated when the heart is dilated or if the stomach is distended. It is desirable to have the bowels thoroughly emptied at least twice daily. Calomel is usually given at the beginning, and magnesia or a saline laxative as needed from time to time.

5. *Specific Stimulation.—Bacterins.*—Cohen does not make use of these before the fourth or fifth day, and then only in a certain number of cases. He uses them in an endeavour to restrict an alarming spread of lesions, or again in cases in which quinine or digitalis has not manifested its influence. Cohen has not much to say in support of this line of treatment. His chief resort to specific stimulation is in instances of markedly delayed resolution. Here success has been invariable. He gives 50 million killed bacteria, and increases the dose by that amount at each subsequent injection, at three, five, or seven-day intervals, according to the reactions observed. Injections are also useful in delayed convalescence from various causes.

6. *Ancillary Measures.*—Creosotal is a useful adjuvant in a minority of cases showing marked pulmonary symptoms—cough, expectoration, and the like. It is also valuable when the temperature is intermittent, and for those who show an idiosyncrasy to quinine. It is usually given in doses of 20 minims at intervals of two or three hours. Of colloidal iodine and silver he has nothing more to say than that they are at present being given a trial.

A careful disinfectant toilet of the upper air-passages, nose as well as throat, is useful in all cases, imperative in many. Spraying or swabbing or both may be employed according to the indications. Among special agents that may be chosen for the purpose are solutions of essential oils in phenolated liquid petrolatum, alkaline detergent washes, solutions of various peroxides, argyrol, and solution of iodine, potassium iodide, and phenol in glycerine. The good effect is well

kept up by continuous inhalation of creosote, or ethyl iodide, or both (with perhaps the addition of chloroform and eucalyptol or terebene), by means of the Yeo (perforated zinc mask) respirator. An adequate supply of fresh air, preferably in an open space, such as a roof or a porch, but with all necessary precautions to prevent chilling of the surface of the body, due attention to nourishment and to the evacuations, and all the other details of good nursing, including watchfulness, almost go "without saying."

NEW BOOKS.

Post Mortem Examinations. By W. S. Wadsworth, M.D. Pp. 598. With 304 Illustrations. Philadelphia and London: W. B. Saunders Co. 1916. Price 25s. net.

In his preface the author states it as his opinion that the methods of approaching the problems of life at the post-mortem examination are in need of vast improvement; that too much stress is usually placed on minor matters, and on obtaining material for subsequent histological examination; and he pleads that the human body should be studied more as a harmonised whole. To assist in a return to this "broader reasonableness" he has brought together some of the results of observations, thinking, and reading, associated with the study of more than four thousand post-mortems.

Dr. Wadsworth is coroner's physician of Philadelphia, and this position, as well as the experience gained by him in it, entitle his opinions to respect and claim our careful consideration of them.

As was to be expected, the book deals principally with the medico-legal aspects of post-mortem work, and chiefly in the light of the requirements of the laws of the United States. The morbid anatomy of lesions does not, as a rule, receive descriptions sufficiently full for the student, or for the medical practitioner. Hence this is a book more suited to the man who is familiar with civil and legal post-mortem work, and who knows his anatomy. To one in this position the remarks of the author on physiological and chemical problems, and even his philosophical speculations—though we are sorry to find these sometimes decidedly cryptic—may give new ideas and open up new aspects of his work. Despite the severe animadversions of the author, we have found that most serious pathologists are not simply "tissue collectors," but are at one in finding that hardly a single examination fails to display something highly interesting, instructive, and even novel.

Part I. is occupied with preliminary observations, and with a discussion of the phenomena of death. Part II. discusses mortuaries, general technique, and instruments: the number of these last described and illustrated tending to give the uninitiated the impression that a huge armamentarium is required. Part III. deals with the examination of the body, general and special, and the text follows the lines laid down by the author in his preface. Part IV. describes special conditions and lesions. This is the weakest part of the book. Part V.,

dealing with medico-legal post-mortems, is the best part, and is thoroughly to be recommended. The last part (VI.) gives useful hints as to photographing morbid conditions and specimens, the repair of the body, conclusions to draw from post-mortem examinations, and gives details of weights and measurements.

Bearing in mind the author's intentions in writing the book, it would be perhaps captious criticism to consider too minutely many points in pathology with which we are not in agreement with him; but we must say that when any author disagrees with the technical methods or conclusions of other authors, we are entitled to expect that he shall describe his alternative methods or conclusions in a manner which can be easily followed and applied. We should not have to pull up at the end of a sentence and ask, "What does the author mean?"

For example, speaking of the liver, Dr. Wadsworth writes: "The ligaments vary with the other fibre-containing structures of the body; and if they vary with the fibrosis in the liver the conclusions are entirely different from when they vary differently from the internal fibrous structure" (p. 336). Again: "A dilated organ may also be distended or contracted so that the degree of the lesion may be misjudged" (p. 437). Surely these sentences could have been put more clearly.

We do not quite see how the following motion can be carrried out: "That most useful motion for which no received name is common in which the knife is made to *vibrate* through a short distance with a *firm hold* and *steady* directed progress." The italics are ours.

Many more or less vague and unorthodox statements might be quoted which any author is perfectly within his rights in making provided he gives his reasons for making them: for example, "stasis that very common cause of inflammatory processes" (p. 294): "'infiltration,' a most unwarrantable retention of the ignorance of bygone days" (p. 436); "emboli, formed by coagulation or *other* disorganisation" (p. 440); "a case of marked auto-intoxication from congenital malnutrition" (p. 340); "certain toxic substances . . . . tend to fibrin deposit (fibroid phthisis)" (p. 291); and so on.

Some of the author's expressions are strong and sometimes amusing; for example, "the metaphysical vagaries of modern science," "the archaic crudities of anatomic nomenclature," "medicated (*sic*) pseudopsychology," "the narrow bacteriologist," "the pangermic hordes." The last is very apposite in these days when pangermanism has been so much discussed.

But, discounting all these criticisms and many more we might advance, we still think that, regarded from the author's own point of view, this book has been written on sensible lines, and that it is one which all those of us who have to teach pathology and to perform post-

mortem examinations should read carefully. His earnestness is shown in one paragraph to which we wholly subscribe, and with which we may conclude this notice: "It is quite as bad to over value some striking change as it is to ignore some apparently insignificant but really potent one" (p. 383).

Nerve Injuries and their Treatment. By PURVES STEWART and ARTHUR EVANS. Pp. xii. + 208. With 97 Illustrations. London: Henry Frowde and Hodder & Stoughton. 1916. Price 8s. 6d. net.

THERE was great need for such a small guide to the subject of nerve injuries as that now before us, and it could not have been supplied by more competent hands than Colonel Purves Stewart and Captain Arthur Evans. Although not entirely devoted to those forms of injury incident to warfare, its special value at the present time lies in the guidance it affords to the diagnosis and treatment of the nerve lesions all too common in our war hospitals. The clinical cases cited and the illustrations selected show that recent experiences in military surgery were the standpoint from which the authors viewed the subject. The crisp and purpose-like manner in which the subject is treated is peculiarly appropriate to the requirements of those engaged in military hospitals, who have only time to master the essentials for quickly recognising the existence and extent of nerve lesions and of determining the appropriate treatment. The first chapter serves to refresh the memory regarding the structure and functions of the peripheral nerves, and the changes which occur after division and during recovery. A glance at Fig. 2 clears up in a moment the whole mystery of the three main classes of sensibility. The second chapter, on "Methods of Examination," is also to the point and avoids all the refinements of the neurological specialist. The different lesions met with, their mode of production, and the conditions which simulate injury of a peripheral nerve, are briefly dealt with—the last-named section being of exceptional value in war work. The general prognosis is dealt with in Chapter V., and the next chapter is devoted to treatment. Emphasis is laid on the importance of treating the muscles and tendons while the nerve is undergoing repair, so that they will be in a condition to react to the impulses when they arrive—a point which is too often neglected in practice. The rest of the volume is taken up with the consideration of lesions of individual nerves and is admirable in every respect. A word of praise is due in respect of the illustrations, which are so numerous and so carefully selected, and they are almost as instructive as the text. We congratulate the authors on having condensed their subject in such a practical way, and commend this work to all who are responsible for the care of the wounded.

Nervous Disorders of Women. By Bernard Hollander, M.D. Pp. iv. + 215. London: Kegan Paul, Trench, Trübner & Co., Ltd. 1916. Price 3s. 6d. net. Also uniform with above, *Nervous Disorders of Men*, and *Abnormal Children*, by same Author.

These three volumes are simply pieces of bookmaking; they are not bad, but it is difficult to see why they were written. The *Nervous Disorders of Women* and the *Nervous Disorders of Men* necessarily overlap to some extent, with the result that not a few passages are almost identical in both. There is nothing very far wrong in either, but also there is nothing that any medical man of common sense and experience could not have put together for himself if he had thought it worth while. A normal layman would find them rather dull, and a nervous subject would be better employed otherwise than in reading aloud his symptoms, while most medical men would read them without learning anything new. Dr. Hollander is a strong advocate of suggestion in treatment, but has really nothing fresh to say about it. Psycho analysis is shortly referred to. He has evidently been more fortunate than most of us in being able to say (*Women*, p. 74): "I have never experienced any difficulty in inducing sleep [in insomnia] without the aid of drugs, even in patients who suffered from acute pains due to organic disease": and again, of women drinkers (p. 144): "If a patient is willing to be treated there is as a rule no difficulty in curing her." He will, we believe, be led by more extended experience to modify both these statements.

Abnormal Children covers less familiar ground. It is a very superficial account of a difficult subject, and consists to a large extent of platitudes about education. Unlike its two companions this volume is illustrated, but unfortunately the illustrations are of the same mediocre quality as the text. On Plate IV. a photograph is described as a "type of imbecile." It suspiciously resembles a case of achondroplasia. One of the "precocious infants" on Plate IX. might be a Mongol (which, by the way, is not depicted elsewhere), but the photograph is not sufficiently clear to make this certain. Plate IX. is surely a joke. It depicts the "business type," the "student type," and the "naval or military type" of child: the examples selected to illustrate these so-called "types" are youngsters not apparently more than 10 years of age. The characteristics of the naval and military type would appear to be a stand-up collar and a black tie!

The Mentally Defective Child. By Meredith Young, M.D., D.P.H. Pp. ix. + 140. London: Lewis. 1916. Price 3s. 6d. net.

This little book has been written specially for school teachers and others interested in the educational treatment and after-care of

mentally defective school children. The author scarcely refers to etiology, pathology, or sociology in relation to mental deficiency, but describes clearly and in simple language the method of examining a defective child, the chief types of mental deficiency, the use of the Binet-Simon tests, the means of educating the feeble-minded, the requirements for special schools, and other subjects of importance to teachers. A long Appendix contains an account of legislation affecting the mentally defective and copies of the Board of Education schedules. The book is not at all original in the sense in which, for example, Binet and Simon's *Mentally Defective Children* is original, but it may be recommended to those for whom it is intended as a decidedly useful introduction to an important subject. Perhaps before a second edition is called for the author may find time to reconsider his opinion that mental deficiency may be caused by a primative union of the cranial sutures, and his statement that idioglossia is practically identical with lolling. His idea that absence of the normal sense in certain cases (p. 48) makes punishment illogical can scarcely be accepted. Kittens are not usually credited with a "moral sense," yet smacking is the popular and successful method of training them to cleanly habits. It is true that the "moral imbecile" cannot be cured in the same way, but the explanation must be found elsewhere than in absence of the "moral sense."

NEW EDITIONS.

The Essentials of Histology. By Sir EDWARD A. SCHAFER, M.D., etc. Tenth Edition. Pp. xi. + 563. London: Longmans. 1916. Price 10s. 6d. net.

WE have pleasure in welcoming the tenth edition of this popular text-book. It is considerably larger than its predecessors, and a number of new illustrations have been added. It is in every way a most satisfactory text-book, but in several directions it contains a good deal more than might be expected in a students' handbook. Notably full are the descriptions of cell structure, the blood, and the nervous system. We notice that a half-page illustration of Gower's haemocytometer has been added. This may be because it is of British manufacture, but no one who has used the Thoma-Zeiss instrument would willingly use Gower's, and no one who has used Bürker's, which is not mentioned, would prefer to use either of the others.

Probably by an oversight we are told on page 43 that before the middle of intra-uterine life the erythroblasts are confined to the marrow. As a matter of fact they are found in the liver till at least the time of birth.

Sir Edward Schäfer urges many facts which favour his view that the erythrocytes consist of fluid contained in a membrane, but the many difficulties in explaining polychromasia and other pathological phenomena on this hypothesis are not considered. On page 286 there is a reference to Lesson XVII. which should read XV.

The author, Messrs. Longmans, and the Darien Press are all to be congratulated on the appearance of this handsome and useful volume.

Mentally Deficient Children. By G. E. SHUTTLEWORTH, M.D., and W. A. POTTS, M.D. Fourth Edition. Pp. xix. + 284, with 20 Plates. London: Lewis & Co. 1916. Price 7s. 6d. net.

THIS book is such an old friend that it is scarcely necessary to do more than announce the appearance of the fourth edition. The chief additions to the text, which has been thoroughly revised, are an exposition of the Mental Deficiency Act and a chapter on the psychopathies of puberty and adolescence. In the latter Kraepelin's views concerning dementia praecox are set forth in some detail. The Appendices contain a great deal of detailed information in a convenient form for reference.

The Purpose of Education. By St. George Lane Fox Pitt. New Edition. Pp. xxviii. + 144. Cambridge University Press. 1916. Price 2s. 6d. net.

This is a short but suggestive essay, largely Herbartian in spirit, which deals with the purpose of education on the lines of the text "You cannot live by bread alone." The author discusses the facts of consciousness as understood by modern psychologists: human personality: and the spiritual nature of man. He emphasises the educational value of high ideals, and tells us much regarding the formation of mental "complexes" and the part they may play in subordinating our lower to our higher nature. The object of the book is stated to be the application of the results of experimental psychology to the elucidation of educational problems, and those who are interested in education will find that the book will repay perusal. The author writes in a condensed style which demands close attention, and we agree with those critics who think ordinary people would have been helped by a more free use of illustration, rather than with the author, who thinks he may expect those who read the book at all to be willing to take the trouble to understand it. The author has, however, been at some pains to meet the views of the critics of his first edition, which appeared in 1913.

NOTES ON BOOKS.

THE bulk of the *Medical Who's Who* (the Fulton Manders Publishing Co., price 10s. 6d. net.) grows apace. In four years it has increased from 300 to 1200 pages. It therefore includes particulars of a very considerable proportion of medical practitioners in the United Kingdom, and has outgrown the objection that it is merely a selected list of doctors. A melancholy interest attaches to the obituary for 1915, which shows in a striking way the yearly wastage in the profession. No fewer than ninety-seven of the names in this list are starred to indicate that they have died on service between January 1915 and the end of February 1916. Unfortunately a considerable number will have to be added in the next issue. A useful list of medical and nursing periodicals is included.

Pye's Surgical Handicraft (John Wright & Sons, Ltd., price 15s. net) is one of those books that no longer call for a detailed review. It has now reached its seventh edition, and under the editorship of Mr. W. H. Clayton-Greene has been brought up to date. A chapter on X rays has been contributed by Dr. Harrison Orton. This is a book which should be in the hands of every house-surgeon.

There is no diminution in the educational value of the *Clinics of John B. Murphy* at Mercy Hospital, Chicago, of which Nos. 5 and 6 of Vol. IV. (W. B. Saunders Co.) are now before us. The range of subjects dealt with in these two numbers is rather wider than in some of their predecessors. The illustrations are numerous and instructive.

We have received the second edition of *The Maintenance of Health in the Tropics*, by W. T. Simpson, C.M.G., M.D. (John Bale, Sons & Danielsson, price 3s. 6d. net.), in which the author has endeavoured to furnish lay readers with some information regarding the maintenance of health in tropical countries. While the book contains much practical advice it is too elementary to be of value to medical men; to the lay reader portions of the text and some of the illustrations—for instance, those of the life-cycle of the malarial parasite and the microscopic features of fleas, ticks, lice, etc.—will prove confusing.

REPORTS, TRANSACTIONS, ETC.—We have received Vol. XX. of the second series of the *Index Catalogue of the Library of the Surgeon-General's Office, United States Army*. It contains the entries "V" to "Waterworks." The accumulating literature on the subject of "Vaccines" is shown by the fact that over ten pages are required to

record the titles of publications on the subject, while almost as many more are devoted to "Vaccine therapy."

The *Transactions of the American Surgical Association* are always welcome as a convenient record of the year's work in the advanced school of surgery in America. The present volume, XXXIII., edited by John F. Erdmann, the recorder of the Association, contains a valuable series of papers. The surgical complications of typhoid fever, operations on the spleen, and other abdominal operations bulk largely. The report of a committee on fractures is included.

The *Transactions of the American Association of Genito-Urinary Surgeons*, Vol. X (Frederick H. Hitchcock, 1915) divides itself naturally into two parts. One of these records the deliberations of the society on a number of the rarer and more abstruse problems of the genito-urinary surgeon. In this part there are notes on cases of a rather unusual character which are naturally of more interest to the specialist than to the general surgeon. One paper of more general interest is that on the "Prognosis of Urethral Structure," which is based on a wide experience of a very large number of cases.

In the second part of the report, however, which records the proceedings of a joint session of the Association with the American Gynecological Society, the papers are such as to interest a very much larger number of people. The majority of them is devoted to the study of chronic infections of the urinary tract. This group of cases is notoriously difficult to diagnose and treat, and it is interesting to note that throughout the writers insist that accurate diagnosis is impossible without resort to the more exact methods of cystoscopy and ureteral catheterisation. An especially instructive paper is one on the significance of vesical symptoms in renal conditions. Cystoscopy and ureteral catheterisation in those cases with vesical symptoms are, in the writer's eyes, as important as is abdominal exploration in a case with abdominal symptoms.

One rather suggestive contribution is that which deals with post-operative infection of the urinary tract, the clinical and pathological types presented, and the treatment suggested. Another paper contains an excellent analysis of the types of pain associated with renal and perinephritic conditions and an estimate of their value as an aid to diagnosis. Emphasis is laid on the necessity of elaborate examination of the urine and of employing other confirmatory aids. There is also a very good monograph on the reno-renal reflex.

Two addresses are reported dealing with calculus in the upper urinary tract, one concerning the tendency to recurrence after operation, and another with the pitfalls associated with diagnosis of renal and ureteral calculus.

No less interesting than the set contributions to the agenda are the discussions which follow them, and in which the views expounded are

criticised and examined by men of great experience in this branch of surgery.

The book contains more than its share of misprints and errors of a minor kind, but no one can read it without being greatly helped to a better understanding of some of the problems of genito-urinary surgery.

EDINBURGH MEDICAL JOURNAL.

EDITORIAL NOTES.

Annus Academicus 1916-1917.

This month we publish our annual Educational Supplement, which contains particulars of the Medical Curriculum in the Scottish Universities and other teaching institutions. In spite of the heavy calls made on the time and energy of the teaching staffs by the public services, it has been found possible to provide a complete course of instruction for students in all the Scottish schools.

In Edinburgh the classes open on Tuesday, 10th October, and continue till Wednesday, 20th December, when the Christmas vacation begins. On Tuesday, 9th January 1917, the classes re-open, and the winter session ends on Friday, 16th March. The summer session begins on Tuesday, 17th April, and closes on Friday, 29th June.

The Admission of Women Students of Medicine to University Classes.

In his inaugural address Principal Sir Alfred Ewing wittily reminded us of the perennial interest which has for several decades attached to the question of admitting women students to the medical classes within the University. During the last few months the subject has again been raised, and without any recrudescence of the heated controversy which has previously accompanied its discussion the matter has been quietly settled in a business-like way by the University Court deciding to open the doors of the medical classes alike to male and female students. The arrangements were facilitated by the friends of the women students offering to hand over to the Court within a year the sum of £4000 towards defraying whatever outlay the University might find it necessary to make to institute the change. This offer the Court accepted, and during the forthcoming winter session the women students will receive instruction under the same conditions as the men. In certain subjects male and female students will attend the systematic

lectures of the professors together; in others, such as Midwifery, Gynecology, and, in the meantime, Materia Medica, the lectures will be duplicated. All the practical classes will be taught to men and women separately save in a few advanced classes. We understand that the Anatomical Department in the New School, Bristo Place, will be reserved for the teaching of women students. The clinical teaching in the Royal Infirmary will be conducted in the wards set apart for women students until further arrangements have been made.

The women students will enjoy the same rights as the men of attending the classes of the extramural lecturers of the School of Medicine of the Royal Colleges.

Lieutenant-Colonel F. M. Caird, R.A.M.C.(T.), Regius Professor of Clinical Surgery, has been appointed a Consulting Surgeon to the Forces in France, with the rank of Temporary Colonel, Army Medical Service.

Lieutenant-Colonel Henry Wade has been awarded the Order of the White Eagle by the King of Serbia, with consent of His Majesty, for meritorious services rendered by him at Gallipoli as captain in the Scottish Horse Brigade Field Ambulance.

CASUALTIES.

Lost at sea, Surgeon Probationer Gerald S. Freeman, R.N.V.R., H.M.S. *Lassoo*, on 13th August, aged 21.

Surgeon Freeman was studying medicine at Aberdeen University, and at the beginning of the war joined the 6th Battalion Gordon Highlanders, afterwards transferring to the Navy.

Died on service, Captain Robert Inglis Binning, I.M.S., in Mesopotamia, on 16th August, aged 31.

Captain Binning was educated at Glasgow University, where he graduated as M.B. and Ch.B. in 1907.

Died on service, Major Norman Ernest Jasper Harding, R.A.M.C., in the Colaba Military Hospital, Bombay, on 10th August, aged 41.

Major Harding was educated at University College, Liverpool, and in the University and Extramural School at Edinburgh, where he graduated M.B. and Ch.B. in 1900. He took the diploma in tropical medicine in the same year. He served as a civil surgeon in the South African War.

KILLED in action, Captain HAROLD HEAFFORD PROUDFOOT, R.A.M.C.

Captain Proudfoot studied in the University of Edinburgh, and graduated M.B., Ch.B. in 1911. He received his commission as lieutenant (temporary) in the R.A.M.C. on 1st May 1915.

DIED of wounds, Captain THOMAS CAMERON HOUSTON, R.A.M.C. (T.), on 25th August.

Captain Houston was educated at Glasgow University, where he graduated as M.B. and Ch.B. in 1915. He joined the Lowland Mounted Brigade Field Ambulance as a lieutenant on 2nd April 1915.

DIED on service, Lieutenant WILLIAM WATT FARRAR, R.A.M.C. (Temp.).

Lieutenant Farrar was educated at the Universities of St. Andrews and Glasgow, graduating M.B. and Ch.B. at Glasgow in 1901.

MEDICAL STUDENTS.

DIED of wounds received on 4th August, Second-Lieutenant FREDERICK B. SANDERSON, R.F.A., aged 27.

Lieutenant Sanderson was educated at Charterhouse, at Caius College, Cambridge, where he graduated as B.A., and at Edinburgh University, where he was studying medicine.

KILLED in action, Second-Lieutenant WALTER EDWIN HAMMOND SANSOM, King's Own Royal Lancaster Regiment, on 17th August, aged 19.

Second-Lieutenant Sansom was studying medicine at the Edinburgh University, and got his commission on 7th December 1914.

Indian Medical Service.

IN view of the present great need for experienced officers in the Indian Medical Service the Secretary of State for India has approved a proposal of the Government of India to retain in their appointments, when it is considered desirable in the public interest, Colonels of the Indian Medical Service after they have completed the ordinary period of tenure, or would in ordinary circumstances be placed on the retired list. In order, however, to obviate the block in promotion which would thereby occur, such officers are regarded as supernumerary in their rank, and promotions are being made in succession.

For similar reasons the following special arrangements have also been made in the case of Lieutenant-Colonels who, under the terms of

the Royal Warrant governing promotion in the Indian Medical Service, may be specially selected for increased pay for ability and merit. The number of officers eligible for this increase of pay is at present fixed at 41. Among such of these as retire in any one year four extra pensions of £100 per annum are distributable subject to certain conditions. Retirements being suspended during the war, the Secretary of State for India has decided as a measure of relief:—(1) that, provided there are a sufficient number of applicants who satisfy the required conditions, these four extra pensions, which would otherwise lapse, shall be allotted in each year, but that payment of the extra pensions shall be held over until the allottees have actually retired; and (2) that such recipients shall be regarded as supernumerary on the list of 41 selected Lieutenant-Colonels, and that promotions to the list shall be made in their place.

The Secretary of State for India has decided that any officer appointed by nomination to the Indian Medical Service during the present suspension of appointments by competitive examination shall be allowed to count:—

1. *As Service for Pension.*—Any service rendered by him during the present war as a medical or combatant officer or in a position usually filled by an officer.
2. *As Service for Promotion.*—Any service rendered after 16th July 1915 which counts for pension under 1.

The reason for confining the service specially counting for promotion to service rendered after 16th July 1915 is that this condition is necessary in order to prevent officers appointed by nomination during the suspension of appointments by competitive examination from obtaining seniority over those previously appointed by competitive examination.—(*Published by request of the Secretary of State for India.*)

THE HISTOLOGY OF DISSEMINATED SCLEROSIS.*

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Part I.

Introduction.

The following study is based upon the detailed histological examination of the nervous organs from nine cases of disseminated sclerosis. This name and its synonyms—multiple sclerosis, insular sclerosis—indicate that the disease they designate has, as its chief anatomical feature, irregular sclerotic patches distributed throughout the central nervous system. Its manifestations, therefore, are protean, and an exact knowledge of its

* The complete account of the important parts of this paper will be found in the *Review of Neurology and Psychiatry*, July 1916, vol. xiv. p. 285 *et seq.*; while the full paper, with clinical history of cases, bibliography, appendix, and 456 figures, has been published in the *Trans. Roy. Soc., Edinb.*, 1916, vol. l., part iii. (No. 18), pp. 517-740.

This research was originated by the late Dr. Alexander Bruce, and undertaken in conjunction with him. Most of the material used had been accumulated by him during the later years of his life. The following preliminary communication, made to the Pathological Society of Great Britain and Ireland in July 1910, represents in brief outline the position which had been reached at the time of Dr. Bruce's lamented death :—

Preliminary Communication on the Pathology of Disseminated Sclerosis. By A. Bruce and J. W. Dawson. Reprinted from *Journ. Path. and Bacteriol.* Cambridge. 1911. Vol. xv. p. 126.)

The plaques in disseminated sclerosis, wherever they are situated, are distributed evidently without any relation to nerve tracts. Their character and appearance suggest a gradual infiltration from some central source into the surrounding or neighbouring tissues. In the cord their tendency is to pass inwards from the meninges in a more or less wedge-shaped form, their relationship to blood-vessels being often difficult or impossible to trace except in the earlier stages. The cerebrum and cerebellum are better adapted to give an idea as to their mode of formation because of the independence of the arterial and venous paths. Within the cerebrum the veins pass towards the wall of the ventricles and the choroid plexus towards the veins of Galen, and have in this way a distribution altogether different from that of the arteries. The same is true of the cerebellum. A study of a series of sections shows that the plaques are deposited in relation to the distribution of the veins and to the walls of the ventricles. An examination of sections of the cerebral

pathological anatomy is essential to the understanding of the various clinical forms of the disease.

The etiology of the disease remains absolutely obscure. The supposition of a selective poison acting through the blood-vessels, which has received the support of most recent investigators, is justified as an hypothesis but remains undemonstrated as a fact. Chief interest has, therefore, centred in the pathological anatomy of the condition, and the object of this study has been to trace, by means of the most recent available specific staining methods, the characteristics of the pathological process and to determine, as far as possible, the histological changes which form its basis.

The earliest pathological studies in disseminated sclerosis were made in chronic cases of the disease, and the lesions described were the sclerotic areas found distributed in the brain and spinal cord. The recognition by later writers of acute stages of the disease and of cases of typical disseminated sclerosis running an

hemispheres strongly suggests that the infiltration is along the lymphatic channels surrounding the veins. Similar conclusions are suggested by study of the sections of the pons, cerebellum, and medulla.

For some years Dr. Bruce's attention had been concentrated on the important part played by the lymphatics in disease processes in the central nervous system, and I had the honour of being associated with him in the investigations which he hoped would throw light on the subject. The earlier results obtained were recorded in the paper "On the Relations of the Lymphatics of the Spinal Cord," and more especially in that entitled "Multiple Neuromata of the Central Nervous System." Since Dr. Bruce's death I have worked up more fully the cases of disseminated sclerosis on which our earlier joint observations were founded, and I have also investigated a considerable mass of new material which has more recently been available. This material includes a case which is of special importance from the fact that a full clinical record taken under Dr. Bruce's personal supervision is in existence, and also on account of its having a fatal issue after a comparatively short course. This more recent work, and especially the observation of the acute case alluded to, brought new facts to light, and necessitated a reconsideration of some of the earlier conclusions.

It is a pleasure to acknowledge my indebtedness to the family of the late Dr. Bruce, not only for the use of the material which belonged to him, but also for contributing to the expense of the research.

During three of the years I have been engaged in this work I held a Carnegie Fellowship, and I desire to thank the Trust for the assistance given and for generous grants towards expenses and towards the cost of illustration. My thanks are also due to the Committee of the Royal College of Physicians' Laboratory, Edinburgh, for the facilities afforded for the research; to Professor Ritchie, the Superintendent of the Laboratory, for his sympathetic interest and criticism; and to Dr. Ninian Bruce for much help throughout the course of the investigation.

acute course has caused me to direct special attention to the early changes and their relation to the development of the chronic sclerotic areas. In spite of the large number of works on the subject of disseminated sclerosis, its etiology and pathology present many problems for future elucidation. Our knowledge of the histology, especially of the early stages, has not kept pace with our recognition of the early clinical aspects of the disease. It was hoped, therefore, that a study of the earliest lesions, in tracing the *rôle* which falls to the various tissue elements before secondary factors had been introduced, would throw some light upon the nature and origin of the process.

The pathological process may be disseminated through brain, pons, medulla oblongata, and spinal cord, and may produce symptoms of a very diverse character. The possibility of any useful clinical classification is, therefore, very slight, and an anatomical basis has been adopted for the classification of the various clinical types. According to the predominance of the symptoms the disease has been divided clinically into cerebral, spinal, and cerebro-spinal forms, and the different possibilities of anatomical distribution and localisation have led to a similar pathological classification. Probably no cases are purely of one type, and experience has shown that, if sufficiently careful search is made, sclerotic areas will be found in nearly all cases distributed through both brain and cord, though perhaps much more marked in one or other position. As far as we know from the findings in other organs of the body, there is no widespread reaction to the etiological factor, such as is found in acute poliomyelitis. The anatomical findings point to a process localised in the central nervous system.

Before referring to some of the problems met with in the study of disseminated sclerosis, it will be convenient to give an indication of the pathological changes found in a well-marked case of the disease. Williamson (1908) in his text-book gives the following clear description of the main features:—

"The pathological examination of the nervous system reveals patches of sclerosis scattered about in the most irregular manner in the brain, pons, medulla, and spinal cord. Both white and grey matter may be affected, though the former is more frequently the seat of the disease. The patches are in some cases grey in colour and sharply defined; in others greyish-white and less sharply defined. Large patches in the cord, medulla, and pons may extend over the greater part of the transverse section.

The largest patches are seen in the white matter of the brain. The older patches are firmer than the normal substance of the brain or cord, but recent patches are occasionally seen which are gelatinous and softer than the normal tissues. The chief macroscopical characters of the lesions are their insular character, their irregular dissemination, the absence of secondary ascending and descending sclerosis in most cases. This feature—the absence of secondary degeneration—sharply distinguishes disseminated sclerosis of the spinal cord from other multiple lesions, such as disseminated myelitis and multiple syphilitic lesions.

"Under the microscope a striking feature of many patches is their sharply-defined nature. This feature is well seen in the sections stained according to Weigert's method. Examination under a high power shows that medullated nerve fibres are generally absent in the sclerotic patches; but sometimes at the border of a patch there is a zone in which the medullated fibres are present, though scanty. At the periphery of some patches compound granular cells are found, along with indications that the morbid process is still active. In the patches of sclerosis the axis-cylinders of the nerve fibres are very often present though the medullated sheaths have disappeared. The ganglion cells of the grey matter escape degeneration for a long period in the diseased patch; but at a very advanced stage they, like the axis-cylinders, may finally disappear. The neuroglia connective tissue in a diseased patch is often greatly increased, and converted into a dense fibrous tissue. Spider cells may be present, but the neuroglia is not richer in nuclei than in the normal condition, In other cases the neuroglia, though increased in amount, has an amorphous or homogeneous appearance under the microscope. In some patches the nerve fibres have degenerated, and spaces are left in the neuroglia from which the true nerve elements have disappeared, but the connective tissue itself has not proliferated. Compound granular cells are often present, especially at the periphery of a patch. They are most numerous in recent patches, but may be absent in old patches. Often there are large epithelial-like cells in cavities near blood-vessels. The walls of the blood-vessels in some cases appear normal; in some cases they are thickened and hyaline, or present evidence of endo- or peri-arteritis; in other cases (in recent patches) the peri-vascular lymph sheaths are filled with round cells, compound granular cells, and fat globules. Often a large altered blood-vessel is found in the centre of a patch of sclerosis.

"Four forms of sclerosed patches may therefore be met with—(1) Patches in which nerve fibres have degenerated, leaving sieve-like small cavities in the neuroglia, whilst the neuroglia connective tissue itself has increased very little; (2) patches in which there is marked proliferation of the neuroglia along with degeneration of nerve fibres; (3) patches presenting diffuse proliferation of neuroglia, whilst the nerve fibres and cells persist; (4) recent patches presenting changes very similar to those of cerebral softening—products of nerve degeneration and myelin drops, fat granular cells, and distension of the peri-vascular sheath of the blood-vessels with round cells. A point of importance is the presence of patches in various stages of development in the same case; some patches presenting firm sclerosis, whilst others are soft and recent, and may resemble ordinary softening."

A brief reference may now be made to some of the main problems met with in the study of the pathology and pathogenesis of disseminated sclerosis. Recent works show how widely views differ regarding these, and how vague and confused are the issues placed before the reader. One reason for this seems to lie in the absence of any distinction being drawn between the question of the nature and that of the origin of the process. These are undoubtedly the two most important problems—(1) What is the nature of the process underlying disseminated sclerosis? and (2) where has it its origin, *i.e.* in which structural element of the nervous tissue does it take its rise?

An attempt to answer the first of these questions has led many writers to a diffuse discussion regarding the distinction between inflammatory and non-inflammatory processes in the central nervous system. The views as to what constitutes true inflammation are nowhere so conflicting as when the term is used in connection with the central nervous system, and in the case of no other organ are the conceptions of different writers as to the relation of inflammation to pure degeneration so fundamentally opposed. The nature of chronic inflammation also is again nowhere so obscure as when the term is used in connection with the central nervous system. Schmaus (1903) has summarised the inflammatory process in the central nervous system under the general conception of a reaction-process, which may express itself by an increase in the vital activity of any of the tissue elements, and, for the purposes of this paper, the term is used in this sense.

Further, the pathological anatomy of disseminated sclerosis

bears no analogy to any other known pathological process in the body. We know, *e.g.*, of no process in other organs in which the relative integrity of the specific functioning parenchymatous tissue is associated with the enormous increase of the interstitial tissue occurring in definite circumscribed areas.

Again, experimental investigation has as yet thrown little light on this question: it has proved only that disseminated areas of myelitis may result in a reparative growth of neuroglia, but it has not proved that areas of disseminated sclerosis proceed from an acute myelitis.

The attempt to define more clearly the nature of the process is rendered more difficult, therefore, by the difficulty of defining the term inflammation in the central nervous system, by the absence of analogies from the pathology of other organs, and by the absence of results realised from experimental investigations.

Two views in particular have been advanced to explain the nature of the process. Byrom Bramwell (1904) has succinctly summarised them thus—"(1) That the sclerotic lesions are the result of some irritant which is distributed through the nerve centres by the blood-vessels. (2) That the disease is due to some developmental or congenital defect of the neuroglial or nervous tissue (perhaps similar to, or analogous to, the gliomatosis in cases of syringomyelia), which renders it more vulnerable or liable to be affected by irritation than the neuroglial or nervous tissue of the normal individual."

These views may be termed the exogenous and endogenous theories, using these words in their strictest meaning. The former ascribes to disseminated sclerosis an inflammatory process as its basis, and most observers accept the inflammatory nature of the process in some form. The latter view, strongly advocated by Strümpell and supported by Müller in his monograph, looks upon disseminated sclerosis as a disease independent of external factors, except as "agents provocateurs." Müller distinguishes between true or primary disseminated sclerosis, a primary glia formation due to malformation of the glia—a disease *sui generis*—and secondary disseminated sclerosis, a myelitic form, one of a community of allied diseases. This standpoint, that a uniform explanation of all cases of disseminated sclerosis cannot be given, is taken up by many recent writers. Others, however, cannot recognise the existence of an acute or secondary disseminated sclerosis which differs in its evolution from the chronic forms, but whose pathological anatomy is yet almost identical.

Concerning the origin, as concerning the nature of the process, the views are no less divergent. Supporters of the endogenous theory see in disseminated sclerosis a multiple gliosis whose origin is, naturally, in the neuroglia tissue. Supporters of the inflammatory nature of the diseased process are, however, widely divided in their views regarding its origin. In a sclerotic patch changes occur in the three separate structural elements of the tissue—(1) The nervous elements—the myelin sheath of the nerve fibre; (2) the interstitial tissue—the glia; and (3) the blood-vessels. The differing views are related to these three components, and according to the anatomical change most in evidence, writers have ascribed the origin of the process to a primary parenchymatous change, a primary interstitial process, or a primary vessel alteration. We must undoubtedly differentiate three groups of changes; and the question constantly arises, which is primary, and are the others secondary, *i.e.* are they so related as to be cause and effect, or are they together due to the simultaneous action of the etiological factor? Many neuro-pathologists believe that here, as elsewhere, it is immaterial whether the reaction is discernible first in the parenchyma, or interstitial tissue, or vessels. It may be assumed that there are individual factors which, through the reaction of the tissues upon the unknown, though probably toxic, stimulation of the three components, determine the anatomical picture, allowing in one case one component, and in another case another component, to come to the front.

It is, therefore, clear that amongst the questions raised in any discussion regarding the nature and origin of disseminated sclerosis are the following:—Its relation to acute, subacute, and chronic inflammatory processes in the central nervous system; and, further, its relation to the different processes, *e.g.* diffuse cerebro-spinal syphilis and diffuse arteriosclerosis, which have, as their terminal product, a sclerosed area.

In addition to its unexplained cause, nature, and origin, numerous other problems are met with. Disseminated sclerosis has been attributed to a variety of causes, some of which will be considered later, but, as has been stated previously, the etiology remains unexplained. A study of the cerebro-spinal fluid in disseminated sclerosis has as yet thrown little light on the disease, but investigations along this line have not been extensive. No culture has been obtained from the fluid, and such organisms as have been found in brain and cord sections must be regarded as having an accidental and not a causal relation to the disease.

In the absence of any specific organism the morbid changes have been attributed to the action of a toxin. This hypothetical toxin has not been isolated, but it is suggested that it forms either in the body, possibly in the course of an infective disease, or is brought there from outside of it. For the distribution of this infective agent we have three paths—the blood-vessels, the lymph-vessels, and the central canal of the spinal cord. In view of the importance of the lymphatics of the central nervous system in the distribution of infective agents, and the distinction, emphasised in numerous recent works, between hæmatogenous and lymphogenous infection of the central nervous system, the histological data which give any aid in understanding this problem of the path of infection will be discussed. Amongst the further questions raised by this consideration will be the following:—Why and how the toxin becomes circumscribed; and why, if it is carried by the blood-channel, it does not spread diffusely but should act arbitrarily on detached smaller blood-vessels or a portion of the distribution of such a blood-vessel.

Further, clinically, the course of the disease is marked by remissions and exacerbations, and, histologically, we find in nearly every case chronic areas side by side with areas of a more acute process. Two questions arise—What relation have these more recent areas to the original pathological process; and, if related, is it that the injurious agent has remained in the body for years or is re-formed there, or is it that the original "noxa" lowered the vitality of certain portions of the nervous tissues, so that, later, other exciting factors overturned the balance of repair and waste, and, in Weigert's sense (*Wegfall von Wachstumhindernissen*), disturbed the equilibrium between parenchyma and supporting tissue, and removed the normal controlling influence which one tissue element of a complex structure normally exercises upon the other tissue elements?

Finally, in this connection must be mentioned one striking characteristic of the picture. Most observers who have had the opportunity of examining cases clinically and anatomically have noted the disparity between the anatomical change and the disturbance of function. From the days of Charcot onwards this has been related to the persistence of the axis-cylinder and the comparative integrity of the ganglion cells in a sclerosed patch, with the consequent absence of secondary degeneration.

Historical.

The lesions in disseminated sclerosis were figured by Cruveilhier in his *Atlas d'anatomie pathologique* (1835-1842), and the condition was first clinically described by Frerichs (1849), Rindfleisch (1863) carefully examined the morbid anatomy, both Cruveilhier and he representing the lesions under the name "grey degeneration." The disease, however, was not generally recognised until Charcot published his famous lectures (1866). Charcot, working at the Salpêtrière, may be said to have given the classical account of the disease, both in its clinical manifestations and its anatomical features. The three cardinal symptoms—intention tremors, nystagmus, and scanning speech—when present, were considered as diagnostic of disseminated sclerosis, and around the three cardinal and essential points of the anatomical picture—absence of myelin sheath, neuroglia proliferation, and changes in the blood-vessels—have been grouped the various theories put forward to account for the origin of the process. It has been named by the French writers "*sclérose en plaques disséminées*," by the Germans "*Herdsklerose*," and the appropriate and expressive term "insular sclerosis" was proposed by Moxon.

The works of Charcot show that, to him, the condition was a distinct morbid entity, and that it bore no relation to the other non-system diseases of the central nervous system. His fundamental observations have influenced all subsequent studies, but it has now come to be recognised that the essential clinical features are the grouping of certain symptoms and their variability, and, pathologically, that the anatomical features, once thought to be distinctive of disseminated sclerosis, may be the final stage of quite different processes. Earlier it has been stated that the two groups of theories put forward to explain the nature of the morbid process may be classified as exogenous and endogenous. As the latter term has come to bear a wider significance than that first attributed to it, it has been thought advisable to refer to it under the name "developmental." The term "exogenous" must be admitted to be synonymous with "inflammatory," using this word in its widest sense, as a reaction process.

In a survey of the literature, therefore, it will be found that writers support the inflammatory, or the developmental, nature of the process, while the views of many recent workers cannot be precisely defined. The inflammatory changes may be primary (*a*) in the neuroglia, (*b*) in the true nervous elements, (*c*) in the blood-vessels, or (*d*) related to disturbances in the lymph circulation. The final etiological factor or

factors which bring the primary change into operation have, as yet, received no satisfactory explanation, but it is agreed that the postulated virus circulates in the blood-vessels or lymphatics and exerts its action primarily on the glia, the myelin sheath of the nerve fibre, or the blood-vessel wall itself.

(1) Inflammatory Nature of the Process.

A. *Primary Change in the Glia.*—According to the writers who support this view the morbid process starts in a formative irritation of the glia, comparable to a chronic interstitial inflammation in other organs, *e.g.* liver or kidney. The thickening of the glia reticulum and the formation of the glia fibrils strangles, as it were, the myelin sheath of the nerve fibres, which gradually diminish in volume and then disappear, leaving the axis-cylinders persisting for a long time.

Charcot and his followers held this view. They regarded the changes in the nerve fibres as secondary to the glia proliferation, and the changes in the blood-vessels as a later and not an essential part of the process. Charcot related the peculiar intention tremor, so characteristic of disseminated sclerosis, to the absence of the myelin sheath from the persisting axis-cylinders. The transmission of voluntary impulses would thus still proceed by means of the denuded axis-cylinders, but it would be carried on irregularly in a broken or jerky manner, and would thus produce the oscillations which disturb the due execution of voluntary movements. It was thought possible also that the naked axis-cylinders might again clothe themselves with myelin, and thus effect a "*restitutio ad integrum.*"

B. *Primary Change in the Parenchyma.*—The writers in this group regard the change in the myelin sheath of the nerve fibre as the primary one. They contend that an infective or toxic "noxa" may act directly and primarily upon the nerve fibre before there is any trace of glia proliferation and without a primary blood-vessel change. The alterations in the vessels are looked upon as accessory phenomena, and the interstitial changes in the glia as dependent upon the parenchymatous degeneration, though it is admitted that the absence of definitely proved areas in the peripheral nerves argues for the presence of glia as essential to the process. Huber (1895) and Redlich (1896) supported this view.

C. *Primary Blood-Vessel Alterations.*—This view, stated in simple terms, is that the chief and essential *rôle* in the process is ascribed to the changes in the vessels; these give rise to an altered nutrition of the surrounding tissue, leading to degeneration of nerve fibres or to an extension of the inflammatory process to the peri-vascular tissue, with subsequent or simultaneous glia proliferation. Rindfleisch (1863) was the first to note the significance of the blood-vessel changes and

the intimate relation of the areas to the vessels, but he gave no suggestion as to the nature of the final cause of the postulated alteration of the vessels, nor of the cause of its special distribution. French writers support chiefly the vascular origin. Amongst those may be mentioned the names of Dejerine and Pierre Marie, the latter emphasising the special relation of disseminated sclerosis to infectious diseases. Williamson thinks that the changes are very suggestive of the presence of some irritating substance in the blood which stimulates the endothelium of the walls of the blood-vessels and of the peri vascular lymphatics, and which causes an extravasation of toxic lymph into the surrounding nerve tissue, with consequent degeneration of the myelin sheath of the nerve fibre, and a reactive interstitial inflammation.

D. *Disturbance in the Lymph Circulation.*—In disseminated sclerosis the lymphatic spaces of the adventitia of the blood vessels are frequently distended and filled either with fluid or with cells, or they are more or less obliterated by dense fibrous tissue. Borst (1897) was the first to bring these changes, which are dependent on a primary disease of the blood-vessels, into causal relationship to the areas of sclerosis. The disturbed lymph circulation expresses itself in characteristic serous infiltration, and the obstruction to the return of the lymph causes a lymph stasis in the area, which leads both to a myelin sheath degeneration and a forcing apart of the meshes of glia on the basis of a hyperlymphosis. When the increased pressure of the lymph is removed the glia undergoes proliferation and invests the persisting axis-cylinders with a more or less dense fibre feltwork.

(2) Processes Underlying Multiple Sclerosis Founded upon a Disturbance of Development.

Schiff and other writers have thought it possible to trace disseminated sclerosis to partial or total absence of myelin sheath formation, for it is known that the appearance of myelin represents a definite stage in the development of the nerve fibre. This myelination sets in at different periods in different tracts, and even in different fibre areas of related tracts, and the naked axis-cylinders might, therefore, be looked upon as incompletely developed nerve fibres. Most supporters of the developmental nature of the process, however, relate it rather to disseminated defects in the glia framework of the central nervous system, referring to it as a "multiple gliosis," comparable with multiple fibromata, lipomata, neuromata, etc. They admit that from this kind of pathogenesis numerous forms of disseminated sclerosis must be excluded, and distinguish, therefore, between primary disseminated sclerosis due to developmental defect in some form, and secondary disseminated sclerosis, the consequence of an acute disseminated myelitis. Strumpell (1896) and Muller (1904) are the strongest supporters of this view.

Methods.

As the significance and interpretation of the observations depend largely upon the judgment passed upon, and the importance attached to, the methods of investigation used, it is necessary to give a brief outline of the methods employed. During the last third of the nineteenth century neuro-pathology laid stress on problems affecting the localisation of nerve fibres in an endeavour to define accurately the position of diseased processes, and to follow out secondary degenerations while almost neglecting the histo-pathology of the diseased processes themselves. Nissl laid stress on the fact that practical neuro-pathology can and must be pursued without regard to definite ideas as to the functional significance of the individual component parts of the tissue. He pointed out that it is sufficient in the first place to collect pathological data regarding definite diseased conditions, and that for that purpose the changes in the supporting tissue—the glia and the vascular connective tissue—may be of greater importance and much more really decisive than the changes in the specific tissue elements—the nerve cells and nerve fibres —which are yet of greater functional importance.

Three groups of staining methods were used :—

1. Methods for fibre-tract degeneration. The Weigert myelin sheath stain and the Marchi method are of special value in giving a topographical distribution of the areas, but they permit of very limited histological observations.

2. The elective staining methods. These render possible the isolated representation of certain tissue constituents, but for this reason do not give a complete histological picture. The methods for representing fibre-tract degeneration must also be regarded as elective staining methods.

3. The diffuse stains. These, on the other hand, *eg.* van Gieson's stain and haematoxylin and eosin, do not sharply distinguish between the individual elements of the central nervous system, yet are of great value in giving an insight into the detailed structural changes, especially when controlled by elective staining methods.

The pieces of tissue were embedded in paraffin, or celloidin, or gum. Adjoining sections were then stained by as many of the available methods as possible, so that, by the aid of differently stained sections, adjoining one another, a reconstruction of the complete histological picture was made possible. The examination of small sections for the recognition of the finer histological details

was supplemented by large brain sections through the whole hemisphere. These sections, stained with Weigert's myelin sheath and a diffuse cell stain, give a very instructive comparative representation of the gyri and their associated sulci, and an idea of the extension of the process.

The following methods have, therefore, been relied upon in the histological analysis of the changes in the several tissue constituents:—For the myelin sheath, Marchi and Weigert's methods supplemented by Heidenhain's iron-haematoxylin stain; for the axis-cylinders, Cajal's and the Bielschowsky-Williamson silver methods; for the neuroglia, Heidenhain's iron-haematoxylin supplemented by van Gieson's stain; and for the cellular constituents, Unna's polychrome, methylene-blue, and the diffuse stains. For use with frozen sections, iron-haematoxylin for the myelin sheath; Bielschowsky's method for axis-cylinders; Lhermitte and Guccione's stains and Ford Robertson's methyl violet stain for glia cells and fibrils; van Gieson's stain and haematoxylin and eosin for the cell elements; and Scharlach R. for fat granule cells.

NOTES ON ACUTE INTESTINAL OBSTRUCTION.

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I.—Obstruction by Bands.

The classical works on intestinal obstruction, such as those of Treves and H. L. Barnard, to mention only two, have dealt with the whole subject so exhaustively that there is little left to be said. Yet as no two cases are exactly alike, the records of individual experiences of different forms of obstruction may still be of interest even if they add to our knowledge little that is of value. The following notes epitomise the records of 24 cases of obstruction due to bands on which I have operated within the last few years.

Clinical Aspects.

Mode of Onset.—The obstruction that results from constriction of the bowel by a band is almost always of the acute type, both as to its mode of onset and its progress. The earliest symptoms are those of peritonism coming on suddenly without previous discom-

fort or feeling of illness, and followed, without appreciable intermission, by those due to interference with the onward passage of the intestinal contents, leading to toxæmia.

Occasionally, however, the onset is less abrupt, and for some days the symptoms fluctuate in their severity so that the seriousness of the condition is not manifest and operative treatment is delayed. This was so in three of my cases:—(1) A woman of 59, who after suffering very severe pain attended with vomiting for some hours, gradually improved, and for four days had symptoms suggestive rather of obstinate constipation than of actual obstruction. On the fifth day the symptoms became much more acute, and she was operated upon. (2) In another woman, aged 50, the severity of the symptoms varied from Sunday till Thursday, the patient at times suffering great pain and vomiting, at others being quite comfortable for hours on end; flatus passed freely, and the bowels were moved by enemata. On Thursday symptoms of complete obstruction suddenly ensued, and operation revealed a band snaring a coil of small intestine near its lower end. (3) In the case of a girl aged 7, in which a loop of bowel was hung over a band formed by a Meckel's diverticulum, the symptoms were not alarming during the first five days of her illness.

Pain.—The initial symptom in all my cases has invariably been acute diffuse pain of a griping character, as a rule centred in the region of the umbilicus, but occasionally referred to one or other iliac fossa. In a few cases there had been antecedent discomfort in the abdomen, with unwonted irregularity in the action of the bowel, but in the great majority the patient was perfectly well until seized by the acute abdominal pain.

In the cases that are acute from the onset, as it is the small intestine that is implicated the pain is continuous and gets progressively worse for three, four, or five days, until toxæmia renders the nervous mechanism of the bowel less sensitive, or perforation occurs and relieves the tension in the distended loop. In none of my cases had perforation occurred before operation, but in three at least the pain became distinctly less severe after a few days, although the strangulation of the gut was becoming more marked.

Vomiting.—Within a few hours of the onset of pain, vomiting, associated with nausea, retching, and eructation, ensues; the vomited matter being at first yellow from admixture of bile, and later dark brown—the so-called stercoraceous vomit. Only in one of my cases was there no vomiting. The patient, a woman aged 60, had all the other symptoms well marked for five days,

and at operation a band was found strangling a loop of small intestine deep in the pelvis. In another woman, aged 24, the vomiting, after lasting for two days, ceased entirely for the next three days, and then began again, the other symptoms not showing the same amelioration; the obstruction was due to a band snaring the lowest part of the ileum. In both cases operation was delayed by this anomalous symptom, but fortunately both patients recovered.

Action of Bowels.—The bowels may act once or twice after the symptoms come on, and flatus may be passed, but in most cases within a few hours the obstruction becomes complete. In one of my cases the patient continued to pass flatus freely for four days after the onset of acute symptoms; in two others enemata relieved the patients of flatus for two days, and in a third case for four days. In none of these cases were the other symptoms less severe than usual.

Examination of Abdomen.—Examination of the abdomen does not reveal any signs characteristic of this particular cause of obstruction, and it is seldom possible to determine the precise site of the constriction. In a few of the cases under consideration localising signs were present and proved useful in determining the site of the incision. A woman aged 40 presented a doughy swelling above the pubes, detectable also on vaginal examination, which corresponded with the seat of the obstruction in the small intestine. In a girl aged 17 a localised tender swelling just below the umbilicus indicated the seat of the obstruction. This corresponded to the site of an adhesion which had caused obstruction four months before, a week after she had been operated upon for acute appendicitis. In another woman, aged 77, a firm mass, mistaken for a malignant tumour, in the left iliac fossa, proved to be the great omentum, rolled up and indurated, and giving attachment to a fibrous band which was snaring the gut. In a man whose abdomen was greatly distended a limited area of dulness to the right of the umbilicus corresponded to the seat of the lesion; and in two women there was excessive tenderness over the strangulated loop of bowel.

Rectal or vaginal examination seldom throws additional light on the diagnosis either as to cause or site. In a case already cited a doughy swelling could be detected on vaginal examination; in a man undue resistance could be felt high up in the rectum on the fourth day of his illness; and in a boy aged 12 a soft, boggy, and very tender swelling was felt towards the right side on rectal

examination. In each of these cases the swelling felt corresponded to the seat of the lesion.

Only in two of my cases has blood been passed per rectum. One was a girl aged 7, in whom the obstruction was due to a Meckel's diverticulum; the blood appeared on the fifth day. The other was a man aged 42, in whom the strangulation was due to an omental band. The bleeding came on at once and continued for three days. He did not suffer from piles.

Previous History.—If a history of some antecedent abdominal illness of an inflammatory nature is forthcoming, or if there is evidence of a previous operation on the abdominal or pelvic organs, the diagnosis of obstruction due to a band is favoured. Six of my patients gave a history of having suffered from symptoms suggestive of tuberculous peritonitis; in one I had previously treated her for this condition. One gave a history of having had appendicitis some years before; in two I had operated for acute appendicitis a year before the obstruction came on, and in a third, a girl of 17, I had operated for acute appendicitis four months before; it is an interesting fact that in this case it had been necessary to open the abdomen a week after the operation for appendicitis on account of acute obstruction due to adhesions involving a loop of the jejunum. In a fourth case the patient had been operated upon for appendicitis 2½ years before.

Operation.—The diagnosis is seldom completed until the abdomen is opened and explored. It is sometimes stated that the escape of blood-stained fluid in opening the peritoneal cavity is strongly suggestive that the cause of the obstruction will be found to be a band, but in only four of my cases is it noted that blood-stained fluid was observed. In quite a number of the cases in which operation was delayed for some days, clear peritoneal fluid was present.

Coils of distended bowel usually present at the wound, and on introducing the fingers similar coils are encountered in every direction. In one case in which the obstruction was situated within a few feet of the duodeno-jejunal junction, coils of contracted gut were met with on opening the abdomen. It is of little use to attempt to trace the dilated loops in the hope of being led to the seat of the obstruction. Without allowing any of the bowel to escape from the wound, the fingers, or if necessary the whole hand, should be introduced and a deliberate search made for some portion of contracted gut. If this proves to be large intestine, as is most often the case, it is traced to the cæcum, and

there the small intestine is picked up and followed till the distended bowel is reached. The meeting-place of the contracted and dilated portions indicates the seat of the obstruction. Gauze packing is then introduced to isolate this area, and the parts are examined to find out the nature of the lesion. The existence of a band is usually quite easy to recognise, but it is often difficult to determine its precise arrangement and attachments. In some cases it is so slender that it snaps across when handled, and the ends recoil and cannot again be discovered: the only remaining evidence of its having been the cause of the obstruction is the passage onwards of the contents of the distended bowel. In other cases it is a strong fibrous cord that requires to be divided with scissors. The cord should be caught with two pairs of forceps and cut between them, so that the loose ends may be traced to their attachments and removed. When structures such as the Fallopian tube, the vermiform appendix, or a Meckel's diverticulum constitute the constricting band, more detailed steps must be taken to deal with them.

When the obstruction has been relieved the strangulated loop of bowel and the coils above and below are examined as to their integrity and viability, on the same lines as in dealing with a strangulated hernia. It would appear that the bowel has a considerable power of regaining its vitality, for in only one of the 18 cases that recovered in this series was it found necessary to resect the strangulated loop, and these were operated on at intervals varying from 2 to 6 days after the onset of acute symptoms, the ages of the patients ranging between 10 and 77 (Table II., p. 247). In another case the viability of the bowel was doubtful, and as the patient was too ill to justify resection, the distended bowel was withdrawn and opened. The artificial anus worked well, and a fortnight later the segment implicated was successfully resected. Of the 6 fatal cases none were resected; in one a loop of distended intestine was withdrawn and a rubber tube inserted into it after the manner of a Witzel's gastrostomy. This served to drain the bowel efficiently, and the patient in addition had several natural motions per rectum before she died ten days after the operation, from a sudden syncopal attack; in another an artificial anus was made the day following operation; in two, post-mortem examination revealed that the bowel had not perforated; in the remaining two no post-mortem examination was made, but the patients only survived the operation a few hours and

appeared to die of the general toxæmia rather than from perforative peritonitis.

After-Treatment.—Most of the patients received a "flatus enema" the morning after the operation, and had olive oil and soap and water enemata for the next two days. On the third or fourth day, according to circumstances, castor oil and calomel (4 grs.) was given. Some patients had small repeated doses of calomel from the first day.

Statistics.

Table I. shows the sex incidence, and the mortality in the cases under consideration.

Table I.

Result.	Males.	Females.	Total.
Recovery . .	3	15	18
Fatal . . .	2	4	6
Total . .	5	19	24

The preponderance of females is striking, and differs from other statistical records. It is not accounted for by the female pelvic organs having furnished the causative band, as there was only one case in which the Fallopian tube caused the strangulation, and one in which an adhesion formed after an operation for ectopic gestation.

The mortality is low compared with most published statistics, but the number of cases is too small to warrant any general conclusion on this point.

Table II. shows the age incidence, and the number of days which elapsed between the onset of acute symptoms and operation. The only comment it calls for is that the condition occurs at all ages. Its comparative infrequence in childhood is doubtless explained by the fact that the patients have not lived long enough to contract the necessary adhesions. This table shows also that delaying to operate increases the mortality.

TABLE II.

	Males		Females.	
	Age.	Days between Onset and Operation.	Age.	Days between Onset and Operation.
Recoveries, 18	16	2	10	3
	34	2	12	5
	42	5	19	7
			24	5
			29	2
			33	3
			33	3
			35	2
			40	2
			50	6
			56	7
			59	4
			60	5
			63	4
			77	5
Fatal, 6	12	7	7	6
	40	4	17	4
			34	4
			40	5

MORBID ANATOMY.

Site.—In the great majority of cases the seat of the obstruction is in the lower part of the abdomen, and the part of the bowel implicated is the small intestine near its lower end. This is what might be expected, as most of the inflammatory conditions that give rise to the adhesions from which fibrous bands develop implicate the organs of the lower abdomen and pelvis. In the female, inflammatory affections of the pelvic organs account for a certain proportion of such adhesions, and in both sexes tuberculous disease resulting in the formation of adhesions is very liable to implicate the lower part of the ileum and the cæcum. It is from the lower coils of the ileum, also, that a Meckel's diverticulum springs, and this structure, acting as a band, is a more common cause of obstruction than the present series of cases would seem to show. Only in one of my cases did the band involve the upper reaches of the bowel—that of a man aged 40, whose illness was exceptionally acute all through, and who died on the fourth day from obstruction of the jejunum a few feet from the duodenum.

This consideration is of practical importance as indicating the seat of the incision, when the presence of a band is suspected to be the cause of obstruction, and no definitely localising symptoms are present. The frequency with which appendicitis and ileo-cæcal tuberculosis cause the adhesions from which fibrous bands originate, would indicate the right iliac fossa as the seat of election for exploring when no more definite indication suggests another site. The more direct the access, the more easily and certainly is the lesion dealt with, and as the seat of obstruction is seldom in the middle line, I have found the lateral incision best.

Nature and Disposition of Bands.—In the majority of the cases in the present series the bands were of the nature of *fibrous cords* due to the stretching and moulding of plastic lymph such as might have resulted from an attack of localised peritonitis. These cords vary in length, some being less than an inch long, while others measured two or three inches. Their thickness was also variable, some being almost thread-like while others were as thick as whip-cord. It is astonishing how slender a band may lead to complete strangulation of a loop of bowel.

As a rule one end of a fibrous band springs from the mesentery of the small intestine. The other end may be attached to the mesentery at another point, to the parietal peritoneum, to the surface of the intestine, or to an adjacent viscus, such as the bladder, the uterus, or the Fallopian tube. All these arrangements were represented in this series. In one case the band arose from the mesentery on one side of the bowel, passed circumferentially round the gut and was attached to the mesentery on the opposite side, thus forming a ring round it. I had no example of a band free at one end lassooing the bowel.

In four of my cases an isolated process of the *great omentum* had formed an adhesion at its free end and constituted the band which strangulated the bowel. (1) In a boy aged 10 a cord-like process from the right border of the omentum had become adherent to the anterior parietal peritoneum just above the bladder, and snared the lower end of the ileum. The appendix was normal. (2) In a man of 40 a similar process of omentum was adherent to the mesentery of the small intestine and to the side of the cæcum, and under it a loop of small intestine, 4 inches long, had slipped and become strangulated. (3) In a man aged 42 the process of omentum passed backward and was attached to the posterior parietal peritoneum, and constricted not only several coils of small intestine, but also the pelvic colon. (4) In a girl aged 12

the lower edge of the omentum was fringe-like and two processes had formed abnormal attachments, one to the mesentery of the small intestine at the site of some tuberculous glands, the other to the fundus of the bladder. The former tag had noosed a coil of small intestine. It is interesting that three of these patients were males.

In one case the *Fallopian tube* and broad ligament formed the band. This patient, aged 56, had a small umbilical hernia, a large reducible inguinal hernia on the right side, and a smaller irreducible inguinal hernia on the left. The irreducibility was due to the Fallopian tube having been carried into the labium, where it became attached and strangulated a loop of small intestine inside the abdomen.

Only in one case, a girl of 17, was a *Meckel's diverticulum* the cause of obstruction. The terminal ligament of the diverticulum was attached to the umbilicus, and a coil of intestine had passed over the band thus formed, and hung across it very much as a wet towel hangs over a clothes-rope.

Mechanism of Strangulation.—It must be confessed that in the course of an operation it is sometimes difficult to determine the exact way in which a band has brought about incarceration of the gut. The critical condition of the patient, the friability of the tissues and the limited field for access, forbid any prolonged and detailed analysis of loops, folds, and knots. The primary objective is the relief of the obstruction, and when this has been effected it is not always easy to reconstruct the lesion and interpret its precise mode of action. The recognition of the band is plain, that it is the cause of the trouble is obvious, but the actual mechanism is frequently obscure and can only be surmised.

In the majority of cases, however, there is no dubiety as to the mechanism. In the present series of cases the commonest was for the band to form an arcade under which a loop of bowel slipped and became caught. This was the state of affairs in more than half of the cases. The length of bowel involved varied from a mere knuckle up to a coil one or two feet in length. In some cases the two limbs of the engaged loop lay parallel, with the mesentery between them flat; in others, the loop along with its mesentery was twisted in the form of a volvulus. This twisting of the mesentery aggravates the effect of the compression of the band by interfering with the blood-supply of the ensnared coil.

The following short notes will serve to illustrate some of the other ways in which a band may occlude the intestine and bring

about obstruction. They do not, of course, by any means exhaust the possibilities.

1. In a woman aged 34, who had been two days ill, a short, broad band was found attached at one end over a calcified gland in the mesentery, and at the other to the convex border of the immediately adjacent small intestine. The traction of the band had twisted the bowel on its long axis, folding it over on itself to such an extent as to occlude it. Fatal.

2. A somewhat similar mechanism explained the obstruction in a woman aged 33. The band sprang from the mesentery and was attached to the distal side of an adjacent coil in such a way that its contraction folded the bowel over on itself and occluded it. Recovery.

3. An unusual mode of strangulation was found in a woman aged 24, where a fibrous cord arose from a calcified gland in the mesentery on one side of the bowel, and passed circumferentially across to become attached again to the mesentery at a point directly opposite, thus forming a ring round the gut, about three inches from the ileo-cæcal junction. The symptoms of obstruction came on with less acuteness than in most cases, and varied in severity from day to day, till she was operated upon on the fifth day of her illness. Recovery.

4. In this case, a woman aged 59, the band was attached to the convex border of two adjacent coils of small intestine and an intermediate loop slipped under it and was strangulated. As the ensnared loop became swollen and distended, it exerted traction on the band, which drew together the coils to which it was attached, and so occluded them also. Recovery.

5. The next case illustrates a state of affairs in which a mesenteric band, in addition to ensnaring a loop of small intestine, so dragged upon the portion of bowel to which it was attached as to kink it, and so cause a double obstruction. After division of the band both the kink and the strangulation were relieved. This occurred in a woman aged 60, who recovered.

6. An almost identical condition was found in a girl of 19, save that the kink was on the distal side of the ensnared loop instead of on the proximal side, as in No. 5. This patient also recovered.

7. In another case, a woman aged 77, the loop that happened to pass under a band had its two limbs bound to one another by firm fibrous adhesions which were evidently of long standing. After division of the band these adhesions were not interfered with, yet the patient made a good recovery.

8. An unusual mechanism was accountable for the obstruction in the case of a girl aged 17. Four months previously I had operated on her for acute appendicitis, and a week later had had to reopen the abdomen to relieve an obstruction of the jejunum due to inflammatory adhesions. Subsequent to this it would appear that the convex borders of two pieces of small intestine had become adherent by plastic lymph, and in course of time had been pushed asunder so that the wall of each was drawn out in the form of a short diverticulum. Under the arcade thus formed a loop of bowel slipped and became strangulated. After the adhesions connecting the two diverticula had been separated, each diverticulum was invaginated into its own piece of bowel by Lembert sutures. As the bowel above was greatly distended and its peristaltic power doubtful, a fæcal fistula was established *à la* Witzel. This served to empty the bowel, but the patient died of syncope about ten days after the operation.

9. The only case in which I have recognised *two bands* sharing in the production of obstruction was that of a girl aged 12, in whom one tag of the free border of the great omentum was adherent to the mesentery of the small intestine over some tuberculous glands, and another to the fundus of the bladder. So far as I could determine it was the former—the shorter and firmer—that actually caused the obstruction, although the loop of gut implicated passed under both. Recovery.

10. Reference has already been made to a case of strangulation by a Meckel's diverticulum (p. 249).

ACUTE GENERALISED TUBERCULOSIS.

By A. FERGUS HEWAT, M.B., F.R.C.P.,
Temporary Lieutenant, R.A.M.C.

THE following case, which was under my care at the Royal Victoria Hospital, Netley, presents certain features of clinical and pathological interest.

1. *History.*—The patient, aged 28 years, had 12 years' military service to his credit, part of which time he spent in India. His health was good till early in October, when he developed acute pleurisy with effusion while on active service in France. A few days before his admission to Netley two pints of clear fluid were withdrawn from his right pleural cavity.

2. *Physical Examination on Admission.*—Temperature, 101 ; pulse, 110 per minute ; respirations, 24 per minute. He is flushed,

but well nourished. There is some pain on the right side on deep inspiration. There is no expansion of lung at the right base. The percussion-note is dull from the angle of the scapula downwards on the right side, and there is absence of vocal fremitus. The breath-sounds are freely heard over this dull area. There is a little friction at the upper part of the dull area, and vocal resonance here is ægophonic in type. Apex of left lung has a slightly impaired note with some prolongation of expiration. Heart normal in size and position.

Progress of Case (in brief). — During the first four days of his stay in hospital patient was feverish, the temperature varying between 100° and 103°. He is an excitable and nervous man. On the third day after admission he was ordered aceto-salicylic acid, 10 grs. 4-hourly, and the day following he was much better. Temperature and pulse fell to normal. No fresh signs developed in the lungs.

For the next four weeks his progress was satisfactory and steady. His chest expansion improved, and the dull area at the base of the lung diminished in extent. He then became worse, felt cold, and shivered. His condition suggested a malarial attack, but no parasites were found in the blood film. No further signs developed in his chest, and in a week his temperature, etc., were strictly normal. As the pleurisy appeared to be of a tuberculous nature, he was sent, ten days later, to a "section" hospital, from which he could be transferred back to Netley if a relapse occurred. He remained there for six weeks, and was then returned with a note stating that he had suffered from pyrexia for the last two weeks of his stay there.

4. *State on Re-admission to Netley.*—Temperature, 100·8; pulse, 78 per minute, tension distinctly low; respirations, 24 per minute. He was flushed, and looked exhausted. Complained of frontal headache, and suffered much from sleeplessness. He was irritable, excitable, and complained that the light hurt his eyes.

Lungs.—No signs of acute pulmonary disturbance were present. Dull area at the right base was the same as when he left six weeks ago.

Heart.—Normal.

Abdomen.—Slight tenderness over the appendix region. Bowels constipated. Tongue very dirty. Urine shows presence of a little albumin. During the first two or three days of his re-admission he vomited two or three times. No epigastric tenderness. Temperature irregular, 103·-99°; pulse regular, but dicrotic and

under 80 beats per minute. No cough, and no change in physical signs of lungs. During the next two days pulse and respirations remained below 80 per minute and 20 per minute respectively. Pulse was very markedly dicrotic. Temperature remained high, 102 -103°. Towards the end of this time a slight degree of Kernig's sign was elicited. Knee-jerks were diminished; abdominal and epigastric reflexes were very feeble. Plantar reflex - flexion. He had marked frontal headache, but there was absolutely no stiffness in the posterior muscles of the neck. On the fifth day after his re-admission he became much worse. Circulation began to fail, and basal crepitations were detected in his lungs. Respirations were only 22 per minute, however. Pulse was markedly dicrotic. Towards the end of the day he coughed up a minute quantity of sputum for the first time during the last five days. Knee-jerks absent. Epigastric and abdominal reflexes lost and tâche cerebrale present; Kernig's sign marked; no stiffness of cervical muscles; pupils dilated, no strabismus. Blood did not agglutinate to the typhoid group of organisms, and his faeces and urine were negative to the typhoid group. A differential blood-count showed an increase of polymorphonuclear leucocytes. He wandered mentally, and tried to get out of bed. Lumbar puncture was performed, but no fluid was obtained. He became rapidly worse. The day before his death he was unconscious. He occasionally put his hand to the back of his head, although there was no stiffness in the muscles there. He had retention of urine. Bowels remained constipated to the end, and were only relieved by giving purgatives and enemas. The diagnosis was a little obscure, but a tentative diagnosis of miliary pulmonary tuberculosis following a pleurisy with effusion and terminating with meningeal tuberculosis was made. Fortunately permission for a post-mortem was granted.

5. *Abstract of Post-Mortem Report.* — The cerebral meninges showed typical early tubercles in the usual sites. An early stage of "matting" of the basal meninges was present. The cerebro-spinal fluid was much increased in quantity.

Lungs.—Right.—Recent adhesions at base; no excess of pleural fluid. On section there was some recent fibrous change at the base. Throughout the rest of the lung there was intense congestion and small yellowish tubercles. No bronchitis and no bronchopneumonic arrangement of tubercles.

Left.—Very small scar at apex of upper lobe. On section the same appearance as in the left, but rather more acute. No

bronchitis. No enlarged, congested, or caseating bronchial glands were present.

Heart.—Dilatation of right side.

Diaphragm.—This muscle was completely covered by small tubercles both on thoracic and abdominal surfaces. They were specially marked on the left side in the splenic region.

Spleen.—Congested, slightly enlarged, and showed tubercle-like masses throughout its structure.

Kidneys.—Both are congested and show tubercles scattered throughout.

Liver.—Some degree of cloudy swelling. No enlarged or congested abdominal glands.

Remarks.—The diagnosis of acute miliary pulmonary tuberculosis is frequently a difficult and uncertain problem. Meningeal tuberculosis is a common secondary phenomenon in children, but is not so frequently met with in adults.

This case at first presented the characteristic features of an ordinary pleurisy with effusion of probably tuberculous origin. His febrile attack and slight increase of pulse-rate during his first stay at Netley were the only indications of poor bodily resistance, but against this there was a steady increase in weight. He made good progress for at least six weeks under excellent conditions. Then pyrexia ensued, which during his second stay at Netley showed very little to guide one to the lung as an acute focus of disease. He had no cough, no sputum, except a minute quantity two days before death; this was thrown out before it was examined. His respirations after his first day were 22 per minute or less till the last forty-eight hours, and he had no fresh physical signs in the lungs. His pulse was singularly slow considering his pyrexia, and most markedly dicrotic. The diagnosis of typhoid fever was considered, but in the absence of various classical signs, a negative bacteriological report, and the presence of a definite pleuritic lesion, was ruled out.

The diagnostic points in favour of a meningeal infection were obvious enough towards the end, but the absence of cervical rigidity, strabismus, and irregular pulse-rate was rather puzzling.

The chief pathological interest of the case, apart from the confirmation of the diagnosis, was (1) the very intense infection of tubercle, and (2) the question of the focus of infection. No bronchial, abdominal, or pelvic glands were discovered which might have been the starting-point of infection. The condition appeared to be a very acute blood infection. The acute yellowish tuber-

culous foci in the lungs were perfectly shown. There was no bronchitis and no bronchopneumonic distribution of infection.

It seems possible that the infection may have started at the base of the right lung and that the blood-stream was there infected. The very small scar at the left apex showed no evidence of acute change. The intestines showed no sign of tubercles, but the diaphragm was most virulently infected, which would point to a lymphatic as well as a blood spread.

In conclusion, I wish to express my thanks to Colonel T. J. R. Lucas, C.B., A.M.S., Officer Commanding, Netley Hospital, for permission to publish the above record. I am also indebted to Major Embleton, R.A.M.C., who kindly made the post-mortem examination.

A CASE OF AMŒBIC ABSCESS OF THE LIVER.

By H. L. WATSON WEMYSS, M.D., F.R.C.P.(Ed.),
Medical Officer in Charge, Dalmeny House Hospital.

W. B., aged 27, a private in the Essex Regiment, was admitted to Dalmeny House Hospital on the 19th of February 1916, from the Mediterranean Expeditionary Force, with the following history:—

He landed in Gallipoli in the beginning of August 1915, having received a single inoculation against typhoid fever during the voyage. A fortnight later he began to suffer from diarrhœa, which continued until his return to this country six months afterwards. He remained, however, in fairly good health till about the 10th October 1915, when he had an attack of dysentery. At this time he received two injections of emetine, which only proved of temporary benefit. He was sent to a rest camp for a week, then returning to the firing line. In the beginning of November he went sick, complaining of pain in the upper part of the abdomen. He was now jaundiced, and the diarrhœa continued. He was again sent to a rest camp, and from there was at once shipped off to Giza in Egypt, where he remained for five weeks. During his last week at Giza he became feverish, and as his blood gave a strongly positive Widal reaction, he was sent to the Choubra Typhoid Hospital, Cairo. Here no definite symptoms of typhoid fever were found; the Widal reaction was still present. A blood-culture was entirely negative, and three examinations of the urine and fæces failed to reveal the bacillus

typhosus. He remained at Choubra from 16th December 1915 to 2nd February 1916, when he was sent to England.

He was admitted to Dalmeny House Hospital as a convalescent case of enteric, and complained of diarrhœa and abdominal pain, chiefly localised in the left hypochondrium. The fæces were examined for typhoid and paratyphoid bacilli with negative results. A milk diet was ordered, and daily colon lavage with saline solution, together with bismuth and salol, were prescribed. For the first week the temperature was usually about 99 F. in the morning, rising slightly in the evening. From five to twelve motions were passed daily. At the end of this period the patient felt much better, the temperature became normal, and only three to five motions were passed daily. A slight advance was now made in the diet, but the temperature again became unsteady, rising to 99 F. or 100 F. in the evening. The appetite was very poor. This state continued for about six weeks, *i.e.* till the middle of April; two or three motions were passed daily and were still bacteriologically negative. The blood now gave a positive Widal reaction with typhoid after two hours (1-60). It gave negative reactions with paratyphoid A and B after two hours (1-30). There was progressive loss in weight to the extent of more than 14 lbs.

On the 25th April, eight weeks after admission, the temperature rose to 101 F. at night, and the patient complained of pain on the right side over the liver. Friction was heard over a small area just above the costal margin in the right mid-clavicular line. The leucocytes numbered 14,000 per cubic millimetre. The temperature now became definitely swinging in character, varying from 98 F. in the morning to 101 F. in the evening.

At this stage Major Boyd saw the patient in consultation. The possibility of an hepatic abscess was considered, and shortly afterwards the liver became evident below the costal margin. Posteriorly there was increased dulness, evidently due either to a pleural effusion (empyema) or to an extension upwards of the liver dulness. Exploratory puncture at the lowest level of the pleura was negative. The dulness continued to increase in an upward direction, and exploratory puncture was again carried out posteriorly in the 9th interspace. No result was obtained. The leucocytes rose to 21,000 per cubic millimetre, and a week later to over 28,000 per cubic millimetre, about 80 per cent. of these being polymorphs. The red corpuscles numbered 3,500,000 per cubic

millimetre. The patient was now suffering much pain on breathing or on the slightest movement, and was unable to sleep without narcotics. The liver became increasingly larger both in an upward and downward direction. Below, it could be felt as a resistant mass extending four fingers'-breadth below the costal margin. Posteriorly the dulness reached as high as the 8th rib, where some friction was heard on 7th May. The respirations, which had varied from 22-26 per minute, rose to 32 per minute. The leucocytes were now 21,400, and the red corpuscles 3,500,000 per cubic millimetre. As the patient was in great distress and was obviously losing ground rapidly, on the 9th May, under ether anæsthesia, Major Wallace explored the liver through the 7th interspace in the posterior axillary line with a large syringe and needle. The needle at once went into a large cavity and some chocolate-coloured fluid was withdrawn. A portion of the 8th rib was then resected and a very large abscess cavity opened and evacuated. Four pints of chocolate-coloured material were measured. A portion of the wall of the abscess cavity was removed for examination and a large drainage-tube inserted.

The contents of the abscess cavity gave no growth after 24 hours' incubation, and amœbæ were not found either in the pus or in the portion of the wall removed. Further examinations were made of the discharge on subsequent occasions up to the seventh day after operation, but the results were all negative. The day after the operation emetine was given twice daily in doses of $\frac{1}{4}$ gr.; this was continued for three days, and after an interval of two days $\frac{1}{2}$ gr. were given once daily for three more days. The patient naturally experienced great relief after the operation. The temperature became normal, and remained so. The liver rapidly resumed its normal size. The discharge had practically ceased at the end of a week. The stools became normal in character for the first time for eight months, and the patient's general health improved enormously. Convalescence was quite uneventful. Between the date of operation and the 31st July, when he was discharged from hospital, he gained 29 lbs. in weight.

The case was of interest, firstly, from the diagnostic point of view, because the patient was sent home as convalescent from typhoid fever, so that the apparently slight attack of dysentery which he had several months before was apt to be lost sight of; and secondly, because, notwithstanding the practical certainty of the amœbic origin of the abscess, it proved impossible, even by repeated and most careful examinations, to demonstrate the

presence of amœbæ. Unfortunately the obsession that the case was one of typhoid fever led to failure to examine the fæces for amœbæ at the time the patient was admitted to hospital, and later the administration of emetine may have been partially responsible for this inconclusive result. It may be doubted, with the evidence at our disposal, if the patient ever had enteric at all. Bacteriological reports were invariably negative, and the positive Widal reaction was to be expected after recent inoculation.

RECENT ADVANCES IN MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

W. T. RITCHIE, M.D., EDWIN MATTHEW, M.D., J. D. COMRIE, M.D., AND A. GOODALL, M.D.

Diagnostic Errors in Senile Cases.

Nascher (*Arch. of Diagnosis*, April 1916) holds that if we were to look upon senility as we look upon childhood—as a physiological entity, and not as a pathological state of maturity—we would get a new conception of disease in old age. The principal sources of error in diagnosis in senile cases are considering the senile degenerations as diseases, mistaking the senile degenerations for diseases which they resemble, overlooking disease which is obscured by the manifestations of senility, unreliable history, ill-defined symptoms and atypical diseases, and misinterpretation of signs and symptoms.

Failure to recognise the atonicity and loss of muscular fibres, and the subsequent fibrosis which determines senile arteriosclerosis, is responsible for many futile attempts to cure the condition. It is almost always possible to reduce the high blood-pressure which is one of the symptoms of this condition, but the relief of a symptom does not restore degenerated tissue. In differentiating senile from pathological arteriosclerosis, etiology, symptoms, and results of treatment must be considered. It is primary, comes on late in life, progresses slowly, and is associated with phlebosclerosis, contracted kidneys, and other signs of senility. The blood-pressure follows the formula, age plus 100 in millimetres for the systolic pressure at the patient's age. Iodides will reduce the pressure, but signs of iodism supervene, and the drug must be discontinued, and the pressure rises again to the normal for the patient's age.

Various visceroptoses which are normal in the aged, and are due to

a flaccid diaphragm and wasted abdominal walls, are often treated as diseases. Gastroptosis and dilatation is the normal condition of the senile stomach. The liver sinks, and may reach below the umbilicus. Enteroptosis is usual, and is generally a contributing cause of senile constipation. The principal causes are diminished peristalsis through atony, and waste of the muscular fibres, and diminished intestinal secretion. These ptoses can be relieved by mechanical supports, but they cannot be cured, although they may be altered by surgical means.

Senile emphysema, with its wheezing respiration and general symptoms of impaired aeration, is sometimes treated as a disease. Its progress may be retarded by mechanical supports and living in a dense, dust free atmosphere, but nothing will restore the wasted alveolar septa.

After the patient has been lying on his back a few râles may be heard immediately after sitting up, but not later. They may be due to the opening up of vesicles which had been compressed during recumbency. Cardiac hypertrophy, the usual condition of the heart in old age, is a compensatory condition made necessary by the loss of elasticity of the arteries and the increased peripheral resistance.

Intestinal degeneration, in which peristalsis is diminished through atony and waste of the muscle fibres and lessened intestinal secretion, and evidenced by constipation, should not be treated as a disease. If the individual has a stool without the use of cathartics every second or third day, nothing should be done to produce a daily stool, as this is the normal condition in that person. There may, however, be a stool every day or second day, and yet the stools are delayed, undigested food particles requiring several days to pass through the intestine. This requires treatment.

Senile arthrosclerosis is a prominent example of senile degeneration often mistaken for disease. The ankle is chiefly affected. The joints are not swollen or tender, and the pains lessen after motion, though prolonged motion causes pain and increasing stiffness.

The normal senile kidney is frequently mistaken for chronic interstitial nephritis. The microscopical change is a uniform fibrosis. The diseased kidney shows scattered areas of hyperplasia besides tubal degeneration. A trace of albumen may be found in the case of the senile kidney, but casts and blood are absent.

Senile cardiac myofibrosis is frequently mistaken for a chronic myocarditis. The senile condition is always primary, its antecedents being cardiac hypertrophy and coronary arteriosclerosis. Chronic myocarditis is always secondary. In both the heart becomes irregular after exercise and regular again after rest. In myocarditis the heart becomes stronger after rest. In the senile heart rest does not strengthen it. The response to digitalis is good and often lasting in myocarditis, while in the senile heart its action is slight and ephemeral. Upon autopsy, pigmentation is found in the diseased and not in the senile

heart. The senile liver, which is smaller and firmer than the liver in maturity, is sometimes mistaken for the late stage of cirrhosis. Pleuritic friction sounds may be heard in the aged, simply as the result of drying of the pleural exudate. Slight tremor, a late manifestation of senile involution, is often mistaken for paralysis agitans. It comes on later, and the characteristic gait and facies are absent. Senility may mask the signs of disease. The aged become more tolerant, and when an aged person complains of feeling ill and becomes restless and irritable and sleeps badly, there is some disease present, though it may fail to give rise to clear symptoms. An attempt must be made to disregard the symptoms and signs of senility and look for new manifestations.

A frequent source of error in senile cases is the unreliable history given by the patient. Owing to weakened memory, early ailments may be forgotten and denied. Present symptoms may be exaggerated or fabricated to gain sympathy, or may be suppressed and denied if there is a fear of a painful examination or operation. The infectious diseases are rare, and always grave. The aged may be carriers of infection. Typical temperature curves are seldom found. Pain is frequently absent in diseases which are painful in earlier life. General discomfort and distress may be complained of, but pain is seldom localised. Reflex vomiting is rare, and only some intense irritation of the stomach will cause it. The reflexes are uncertain signs, as they may be altered in either direction in the aged.

The most fruitful source of error in the diagnosis of senile cases is misinterpretation of signs and symptoms. Many of the signs of diabetes and chronic nephritis may occur simply as a senile change. The following signs are commonly misinterpreted:—

Temperature.—The temperature of the aged is low. A temperature of 99° F. in the mouth may be three and a half degrees above normal.

Pulse.—A hard, irregular pulse may occur in arteriosclerosis with myofibrosis, beats being lost in transmission.

Blood-Pressure.—A pressure corresponding to the formula, age plus 100, indicates that the arterial degeneration and the cardiac hypertrophy are progressing normally.

Pain.—This is very uncertain, and anæsthesia may be due to mental impairment.

Eyes.—The pupils are generally contracted, and if the aged person with presbyopia looks at a near object without his glasses, he stares, and has the appearance of suffering pain.

Face.—The face is usually sallow, and there may be areas of passive congestion over the malars, resembling the flushes of pneumonia.

Body.—The skin may look muddy, as the aged may find it difficult to get in and out of a bath.

Heart Impulse.—This may be strong if the intercostal muscles are

wasted, and the only guide to the condition of the heart is the quality of the sounds.

Vertigo.—If due to arteriosclerosis it appears at irregular intervals, and is not affected by light, sound, or position. Many cases are due to gastric disturbance.

Incontinence.—Dribbling of urine and escape of faeces are due to loss of the subconscious mental control, and indicate mental impairment, though the reasoning faculties may appear sound.

Spleen.—This organ is usually greatly atrophied.

Gastric Crises.—Old persons often give a history of pain in the stomach or to the right of that organ, resembling gall-stone colic. In most cases it is due to abdominal arteriosclerosis. It occurs at a much later age than gall-stone colic, and is not accompanied by jaundice or clay-coloured stools.

The Frequency of Unsuspected Syphilis.

McLester (*Journ. Amer. Med. Assoc.*, 24th June 1916) has carried out a Wassermann reaction test as a matter of routine in private practice, and reports on 300 consecutive cases. Practically all the cases were seen in consultation, and thus presented more or less difficult or obscure conditions. Of the 300 patients, 56 gave a positive reaction. It would appear that syphilis can produce almost any disease-complex. Of the 56 positive cases, 22 gave a history of syphilis, while 34 gave no such history either direct or suggestive. The most important group was formed by the neurasthenics. There were 32 patients who, according to present usage, must be diagnosed neurasthenics. Of the 32, 15 gave a positive Wassermann. Beyond this finding there was no demonstrable evidence of disease. Of the 15, only 2 gave a history of syphilis. Unfortunately, the cerebro-spinal fluid was examined in only 5 cases. The question whether these cases are early general paralytics is discussed. Are they paretics of a peculiar type, or are they suffering from a mild, chronic form of cerebro-spinal syphilis? Two conclusions seem warranted from the study: (1) That syphilis is a vastly more important factor in internal medicine than is commonly thought, and (2) that a large number of so-called neurasthenics are infected with syphilis, and possibly present a mild form of cerebro-spinal syphilis.

Trench Fever and its Allies.

During the winter of 1914-15 there was a large number of cases of pain and stiffness in the muscles, which were diagnosed for the most part as "myalgia," and there were others in which the sudden onset of fever with malaise and bronchitis led to the diagnosis of influenza. About the end of April cases of short fever began to occur, which appeared to most medical officers to be a new form of disease. The

numbers affected were very great. It is impossible to give the totals, since they were sent in under many headings. Myalgia and influence were still retained, but pyrexia of unknown origin was the favourite diagnosis. Some were called rheumatic fever, but it was one of the surprises of the campaign that rheumatic fever was hardly ever seen at the time. Herringham did not see more than five cases between October 1914 and October 1915, but it increased in the next six months.

The cases of short fever have occurred literally by the thousand. They diminished to some extent in the winter and autumn of 1915-16, but increased again in the spring of 1916. In the spring of 1915 medical officers were struck by the relapsing nature of the illness. It was this especially that led them to believe that they were dealing with a new complaint, and a few months later descriptions of the condition began to appear in the medical journals. It occurred equally among officers and men, but at first it was seen only among two classes—those who had been at the front, whether in trenches, gun positions, or similar duties, and those attached to field ambulances and field clearing stations to which the patients were conveyed. Afterwards the schools of gunnery seemed to become infected by men sent back from the front.

Herringham points out that nephritis, which is also frequent, though not so common as fever, differs from it in two important respects. It is rare, though not unknown, in officers, and it attacks men at the front, on the lines of communication, and at the base in about equal proportions. Whereas the name "trench fever" has a justification in that the disease appears to be really connected with the conditions at the front, there is no reason whatever for using the term "trench nephritis."

Two types of fever are recognised. In one the temperature rises to 102 or 103 on the second day, has become normal on the fourth day, begins to rise again on the fifth day, and reaches about the same height as before on the sixth day, to fall again on the seventh. In the other, the temperature rises gradually to about the fifth day, and gradually falls to normal. The evening temperature is usually 2 or 3 degrees higher than the morning. The first type of chart may be called the saddle-backed, the latter the hog-backed. The symptoms of the latter are not different from those of the former, but these are themselves, with the exception of the shin pains, so like the general malaise of all fevers, that their occurrence can hardly be said to prove a common infection for the two types. The symptoms vary in different cases and at different times, and are never much more than the common signs of all fevers. The onset may be gradual, but is more frequently sudden. In many cases there is a relapse, but this may be absent. This provides an interesting analogy with the three-day, eight-

day, and twelve-day fever seen in Malta, which all seem to be the same disease, the three-day being a fever of one bout, the eight-day of two bouts, while the twelve-day is a continuous fever. The pulse is nearly always below 100, varying with the temperature. There may be pain in the eyes, the head, the back, the left side, the epigastrium, and the limbs. The most characteristic is that in the lower limbs. It may be the thighs, sometimes about the knees, though not the joint, but the most striking and commonest site is the legs, sometimes the calf, but usually the shins. It often prevents sleep. It is associated with tenderness, and sometimes patients cannot tolerate the weight of the blankets on their limbs. Catarrh is usually absent. Nausea and vomiting are exceptional. Constipation is the rule. Rapid recovery is usual, though exhaustion and tachycardia have been noticed. A very few cases show albuminuria. The spleen is not enlarged. The possibility of enteric, or enteric modified by inoculation, can be fairly easily disproved. The agglutination tests fail, and the bacilli cannot be obtained. The curious relapses are unlike anything seen in influenza. Influenza bacilli cannot be recovered, and catarrhal symptoms are absent.

M'Nee and Renshaw have shown that trench fever can be inoculated by the blood from the sick to the healthy, and, further, that the infective agent is connected with or in the red corpuscles, and is not in the serum.

Hunt and M'Nee consider that the body louse is the means of transmitting the disease in Nature. They regard the incubation period as between 14 and 24 days.

Treatment seems to be of no avail except in the control of symptoms. Morphia generally relieves the pains sufficiently to permit of sleep.

Literature.—Graham, *Lancet*, 25th Sept. 1915. Rankin and Hunt, *Lancet*, 20th Nov. 1915. M'Nee and Renshaw, *Brit. Med. Journ.*, 12th Feb. 1916. Herringham, *Quart. Journ. of Med.*, July 1916. Hunt and M'Nee, *ibid.*

A. G.

SURGERY.

UNDER THE CHARGE OF

J. W. STRUTHERS, F.R.C.S., AND D. P. D. WILKIE, F.R.C.S.

The Treatment of Wounds Involving the Skull.

Cunéo (*La presse médicale*, 6th July 1916) discusses the result of his experience in a series of one hundred cases of gunshot wounds involving the skull. The author's remarks are confined to cases where the projectile has struck tangentially without penetrating the interior of the skull. At Verdun this type of head injury was much the commonest.

In the case of gunshot wounds of the skull, death may be due to the grossness of the lesion or to subsequent infection. Slowly developing intracranial hæmorrhages, such as occur in civil practice, are extremely rare. Extradural hæmorrhage was present in only three of the hundred cases, which were trephined, and even then it was not the predominant feature. The chief aim in operating is to prevent infection of the wound and of the brain and membranes.

Infection is as liable to occur after slight as after severe injuries, and it is therefore important, when the scalp is wounded, to exclude the presence of damage to the skull. In order to establish the presence or absence of a lesion of the skull it is necessary to make a local examination, preferably by excising the edges of the superficial wound in order to expose the deeper parts. The absence of symptoms indicating a lesion of the central nervous system is insufficient evidence to exclude an intracranial lesion.

The least fissure of the bone is sufficient cause for operation, even in the absence of cerebral symptoms. Similarly, if the skull is intact, operation is indicated if the symptoms point to an intracranial lesion. The author has had four cases where there was extensive injury to the inner table whilst the outer surface of the skull was intact.

In the case of extensive injuries of the vault of the skull with loss of cerebral substance it can be said that the trauma has accomplished much of what requires to be done. The surgeon should confine his attention in such cases to paring the edges of the wound and to freshening the edges of the gap in the bone; splinters from the inner table, which often pierce the cortex, should also be carefully removed. There is usually no question in these cases of a formal incision and trephining.

When a wound of the scalp lies over a fissure or localised depressed fracture the exposure required can be obtained by enlarging the wound or by a crucial incision, according to circumstances.

If trephining is necessary the opening should be a small one which can be enlarged as desired with nibbling forceps. Large trephine openings are unnecessary and often harmful.

When the outer table of the skull is fissured, there is nearly always splintering of the inner table, except in the occipital and temporal regions, where the bone is thin, and if fractured at all, the whole thickness is usually involved.

Removal of splinters is easy if the dura is intact, but if the latter is torn it may be difficult to remove all the fragments, and this is best accomplished by gentle curettage of the exposed surfaces.

Occasionally a splinter of bone has to be extracted from a venous sinus which has been pierced: free hæmorrhage is likely to follow, but this can be readily controlled by pressure.

The prognosis, in cases of skull injury, depends mainly on whether

the dura mater is intact or not. If the dura is intact, recovery follows in the great majority of cases, but if it has been penetrated the patient subsequently dies in a large number of cases (60 per cent. mortality in the author's series). The cause of this high mortality is due apparently to exposure of damaged cerebral tissue to surface infection, as the author has observed that incision of the dura, when the cortex is found intact, does not interfere with the patient's recovery even when the dura is left unsutured.

The actual cause of death in cases where the dura is torn is a slowly developing meningo-encephalitis usually coming on after a more or less prolonged period of apyrexia.

Infection rarely spreads from fissured fractures involving the septic cavities at the base of the skull, and this type of injury is rare in gunshot wounds.

Spread of infection from septic wounds of the scalp is favoured by the absence of an efficient defence mechanism in the tissues bounding the subarachnoid space, which shows little tendency to become obliterated, as in the case of surfaces covered by peritoneum.

Cunéo is convinced from his experience at the front that early evacuation of the cases is an important factor in favouring the spread of infection to the meninges. As far as possible, cases of head injury should be retained till the scalp wounds have healed.

The mortality rate of 60 per cent. in the author's series of cases, in which the dura was torn, has led him to try a new method of treatment. In the cases quoted above, associated with the high mortality rate, the author had made no attempt to obtain primary or secondary union of the soft parts but had kept the wounds open and packed. In a subsequent series of five cases the author has deliberately planned his operation, with favourable results, in order to obtain a closed wound. The procedure recommended for this class of case is as follows:—After having repaired and sutured the scalp wound a large flap is reflected, sufficient freely to expose the lesion in the bone. After trephining, damaged cerebral tissue is removed through the opening in the dura. This is usually associated with free haemorrhage, but the latter can be arrested by light packing which, when left in place, occludes the opening in the bone. After forty-eight hours the pack is removed with the assistance of a peroxide spray, care being taken to handle the parts gently in order to avoid re-starting the haemorrhage. The flap finally is replaced and fixed by interrupted sutures. Occasionally, it is impossible to close the original wound completely, after repairing the edges, but the deficiency in the scalp is not necessarily closely related to the gap in the bone.

In a series of five cases, when the surface of the brain was considerably damaged, this procedure proved successful, the patients finally being dismissed cured. The point which the author wishes to emphasise

is the importance, apart from other details in technique, of obtaining a closed wound as soon as possible.

A Two-Stage Operation for Removal of Intracranial Projectiles.

Abadie reports to the Société de Chirurgie (*Rev. de Chir.*, February 1916) a procedure which he has employed successfully in the removal of intracranial foreign bodies resulting from gunshot wounds.

As early removal of a foreign body is important, an X-ray examination should be made as soon as possible in order to confirm the diagnosis. The exact position of the foreign body is then noted by means of a localising compass. If the gap in the skull gives sufficient access, the foreign body can be removed without further delay; if, on the other hand, it is found that the gap in the bone is not sufficiently large, the operation should be conducted in two stages.

The first stage consists in trephining the skull at a point which will allow of suitable access for extraction of the foreign body, and at the same time will permit of the main vessels and cortical centres being avoided. After removal of the trephine circle, an opening is made in the dura mater sufficient to permit a fine pair of forceps to be inserted. The second stage of the operation is postponed for twenty-four or forty-eight hours, when the foreign body is extracted under guidance of the X-ray screen. No anæsthetic is required for this stage.

The advantages claimed for this method are that the foreign body can be removed without troublesome hæmorrhage, that during the extraction manipulations are reduced to a minimum, and that therefore the risk of subsequent infection is diminished. The author has successfully operated with this technique in two cases.

Transplantation of Cartilage.

A short note is given (*Rev. de Chir.*, March 1916) of a paper read by Morestin at the Académie de Médecine on the value of transplantation of cartilage in the repair of deformities, especially of the face, which have resulted from the mutilating wounds so common in modern war. Cartilage has been used in the past for re-formation of the nose, but Morestin has further extended its use to the repair of defects of the skull and facial bones. One advantage of cartilage is that it is more readily tolerated as a graft than most other tissues. The transplant continues to live and undergoes little alteration, there being no tendency for it to be absorbed. A further advantage of cartilage over bone is the ease with which it can be cut into the desired shape. Portions of the sixth, seventh, or eighth costal cartilages can be readily excised, without detriment, for the purposes of transplantation, and if necessary the cartilage can be taken from another

individual. There is no objection to the latter source other than sentimental.

In the case of the skull, the transplantation of cartilage is valuable in filling up large gaps, especially in the frontal bones and if the orbital margin has been destroyed.

In the same way, deformities due to loss of part of the superior maxilla, including the infraorbital margin, or of the malar bone can be corrected. Favourable results are more difficult to attain in the case of the inferior maxilla.

The Technique of Splenectomy.

The indications for splenectomy have recently been extended, and it is desirable therefore to have a definite plan for carrying out the operation. Balfour (*Surg. Gyn. and Obstet.*, July 1916) gives the details of the operation which he performs in the Mayo clinic. Most of the patients operated upon have been cases of chronic disorders of the blood such as splenic anæmia, hæmolytic jaundice, and pernicious anæmia.

Access is obtained through a Bevan incision which splits the left rectus, extending downwards to the level of the umbilicus or lower; the upper end of the incision inclines at an angle inwards towards the middle line in a similar way to the Mayo Robson incision for exposure of the gall-bladder.

The dislocation of the spleen from its position against the diaphragm is the first step in the operation. Diaphragmatic adhesions can usually be safely separated by the fingers. Occasionally, adhesions have to be divided between forceps. Immediately the spleen has been dislocated, a long, hot abdominal pack is carefully arranged in the space formerly occupied by the spleen; the advantage of the pack is twofold, as it serves both to arrest venous oozing and to support the spleen during the further manipulations.

The spleen having been elevated in this way is still connected by the main splenic pedicle with its peritoneal investment, the lieno-renal ligament, the gastro-splenic omentum, and by adventitious adhesions in some cases.

Before tying the splenic pedicle, adhesions as well as the peritoneal attachments should be divided.

The gastro-splenic omentum should be tied in the first instance, in sections, cutting between ligatures as close as possible to the spleen. At the upper edge of this omentum the fundus of the stomach will be found in close relation, and care must be taken to avoid injuring it.

The spleen should now be further mobilised by division of the peritoneal and fibrous coverings of the pedicle. At this stage the tail of the pancreas is in danger, and must be looked for in all cases. Unless

this is done in some cases ligature of the pedicle would be impossible without including a portion of the pancreas.

The splenic pedicle consists of the artery and its accompanying veins. The artery often continues well into the hilus before dividing into its branches. The splenic veins are larger than the artery, but do not bear any constant relation to it, always entering the hilus in two or more branches. The vessels entering the hilus have a fan-shaped arrangement and the veins are very friable. The pedicle may be ligatured *en masse* or in sections, preferably the latter.

The extent to which the spleen can be lifted out of the abdomen is surprising, once the peritoneal and fibrous coverings of the pedicle have been divided, but the friability of the veins must always be remembered.

When it is possible the splenic artery should first be ligated and then the veins. If ligation *en masse* is necessary, on account of the arrangement of the vessels, the two-clamp method is recommended. Two curved clamps are placed on the pedicle, with a third clamp on the spleen side to control return bleeding. A double strand of No. 2 plain catgut is tied, with moderate tension, in the crushed line of the inner clamp, as a partial control, and a second strand is transfixed below the distal forceps as the actual control.

Finally, the large hæmostatic pack is removed, and any bleeding point disclosed is secured by fine catgut on a needle.

In pernicious anæmia few adhesions occur, and the pedicle being small, removal of the organ is usually easy. Splenectomy is definitely contra-indicated during an acute crisis of the disease, or in a period when the patient shows a steady decrease in hæmoglobin and red cells, or in the terminal stages when there are mental torpor, cord changes, or œdema.

The risks of the operation are greatest in splenic anæmia, and the actual operation is also more difficult because of the large size of the spleen, the presence of adhesions, and the special friability of the vessels.

The operation in cases of hæmolytic jaundice is as a rule easy, and there is no special risk. It is, however, most important to avoid operating during an exacerbation of the symptoms. J. M. G.

OBSTETRICS AND GYNECOLOGY.

UNDER THE CHARGE OF

A. H. F. BARBOUR, M.D., AND J. W. BALLANTYNE, M.D.

Spurious Appendicitis of Pregnancy.

Dr. Vautrin writes interestingly (*Ann. de gynec. et d'obstet.*, 1916, 2 s., xii. 177), of what he terms false appendicitis of pregnancy. He has met with five cases of this rare complication of gestation; in all of

them the diagnosis of appendicitis had been based on clinical characters apparently conclusive and precise, and yet in all it was contradicted by the conditions found on operative interference. In the first case the symptoms were so pressing that an operation of urgency had to be performed, and no fine differential diagnosis could be attempted. It turned out to be a gangrenous diverticulitis, and the appendix was absolutely healthy. The operation, which was fortunately undertaken before the peritoneum had become infected, was entirely successful. There were two cases in which a small suppurating dermoid cyst of one ovary was regarded as an appendicitis with a peri-appendicular abscess; operation cleared up the diagnosis in each instance. The fourth case was one of peculiar interest. The patient, a big, strong woman of 28, was suddenly taken ill during the second month of her first pregnancy with sharp pain on the right side of the abdomen, and fainting. A doctor was summoned, and he, noting the small, rapid pulse, the resistance in the abdominal wall on the right side, the frequent vomiting, and the abdominal distension, thought of appendicitis and recommended medical treatment. During the days which followed, syncope and the other symptoms recurred, and when Dr. Vautrin was called to see her he found intense tenderness at MacBurney's point and other physical signs which seemed to point so conclusively to appendicitis on the point of rupture that he opened the abdomen at once by Jalaguier's incision. He then discovered a healthy cæcum and appendix under a collection of blood clot which extended into the right iliac fossa from the right angle of the uterus. After the blood had been removed Dr. Vautrin found a ruptured ampullar tubal pregnancy on the right side. Not wishing to throw the ovary out of action he ligatured the utero-ovarian vessels and put a second ligature on the middle part of the tube and the vessels running parallel to it. The hæmorrhage was thus checked and the abdomen was closed, a drain being left in the iliac fossa for 24 hours. The patient made a good recovery, and the uterine pregnancy which co-existed with the tubal one pursued its course without incident. Dr. Vautrin was of opinion that the two ova in this case were impregnated about the same time, and his reason for leaving the ovary was fear lest its removal, entailing the destruction of the corpus luteum, might cause abortion to take place. The fifth instance of spurious appendicitis was not detected till the puerperium; sudden symptoms led to the opening of the abdomen, when a suppurating ovarian cyst with torsion of its pedicle was found. The lesson to be learned from Dr. Vautrin's cases would appear to be that in the presence of signs pointing to appendicitis in pregnancy one should think also of co-existing tubal pregnancy, of diverticulitis, of a suppurating dermoid cyst of the ovary, and of an ordinary ovarian cyst with a twisted pedicle. One may add that an obstinate constipation ought not to be forgotten.

Post-mortem Cæsarean Section.

Dr. James A. Harrar is quite right when he states (*Amer. Journ. Obstet.*, 1916, lxxiii. 1046) that the sudden death of a pregnant woman undelivered always leads to an embarrassing situation, and it cannot be denied that in many such cases a baby's life is sacrificed when a prompt incision of the abdomen and uterus would rescue it without difficulty. Of course, post-mortem delivery need not necessarily be by Cæsarean section; attempts to extract the child immediately after the mother's death have been made by version or forceps, preceded sometimes by vaginal hysterotomy or manual dilatation, but Dr. Harrar has not found that such attempts are hopeful save in the case of prompt low forceps when the woman has died near the close of the second stage of labour. It is always better to secure the consent of the husband or family to post-mortem delivery if it can be instantly got. But, says Dr. Harrar, if consent is not promptly to be had, or if it is refused and the fœtal heart is still going, it is not only permissible but imperative that the physician immediately do everything in his power to save the child. He adds, and the addition is important, "that the operation of post-mortem Cæsarean section is legal without consent has been shown by C. S. Bacon (1911), who pointed out that the right of the husband to the custody and control of his dead wife's body does not go to the extent of preventing disposal of it as may be necessary to keep it from injuring another human being. The viable fœtus has the same right to live as any other human being. This rule would require a post-mortem delivery in every case when the fœtus of at least the twenty-eighth week of gestation is known to be alive at the time of the mother's death." An associated problem is the legal status of the child born by Cæsarean section after the death of the mother, and the author was told by the head of the legal department of one of New York's largest insurance companies that a child so delivered would share in the distribution of an insurance policy of the mother's for the benefit of her children living at her death.

Dr. Harrar gives the results of the experience gained in the Lying-in Hospital of New York City. In 91,600 pregnancies at or near term there were fifty women who died undelivered, mostly within an hour after admission and some while being taken in; in nineteen of these cases post-mortem delivery was effected. Seven times version was done, all the children being still-born; one child was saved by low forceps, and one by breech extraction; and the remaining ten dead women had the child extracted by Cæsarean section. In the case of the first three of the ten Cæsareans the infants were all still-born; the causes of maternal death were œdema of the lungs in connection with cardiac dilatation, hæmorrhage from placenta prævia, and rupture of the uterus. The fourth mother died from eclampsia in the ambulance,

and twins were extracted alive by the section, although unfortunately both of them died about twenty minutes after birth; it must be stated, however, that a post mortem version had been attempted and had failed. The fifth case registered a success; the mother died from heart failure (mitral stenosis), the fœtal heart was heard after her death, seven minutes later a living child was extracted by the Cæsarean section. It was badly asphyxiated at birth and its heart was as slow as forty per minute, but after forty five minutes it was resuscitated, and it survived. In three other cases (two of broken cardiac compensation and one of eclampsia) the children had beating hearts at the time of extraction, but were not resuscitated. In the case of a woman who died of tubercular meningitis (confirmed at the autopsy) a living child was extracted by the section two minutes after the maternal death; but the infant died six days later from inanition and pneumonia. The tenth patient was under Dr. Harrar's care: the maternal death was from cerebral hæmorrhage and labour had not commenced: the house surgeon who was watching her at the moment of death did the Cæsarean section immediately and delivered a living child who survived. The ultimate result, therefore, in the ten cases, was that two living children left the hospital; in both these cases the section was done promptly, in one instance immediately, and in the other seven minutes after the mother's death. In a case operated on eight minutes after the maternal death, the infant gave a few feeble gasps and then died. The conclusion, therefore, is that the section to be successful must be done at once, and a practical suggestion by the writer is that valuable minutes should not be lost in auscultating for the fœtal heart after the maternal death. Another important hint is that great patience is needed, and long continued efforts at resuscitation of the child: attempts should be persisted in so long as the feeblest flutter of the child's heart can be felt. Any cutting instrument which is at hand may be used for making the section.

Another article on the subject of the post-mortem Cæsarean section is by Mr. George W. Whiteside (*Amer. Journ. Obstet.*, 1916, lxxiii. 1051). It deals with the purely legal questions involved, and is highly technical. A most important point is the matter of responsibility in the cases in which the medical attendant might do Cæsarean section after the death of the mother and fails to do so. Is he guilty of homicide in respect of the unborn child? Mr. Whiteside admits that there is no precedent, but he sums up the matter in the following sentences:—"Inasmuch as our statutes are interpreted in the light of scientific progress, what might not have been considered as culpable negligence half a century ago, to-day would be considered as gross culpable negligence. This principle is well founded in precedent and has found expression in cases where safety devices of recent invention readily available but unheard of and unknown half a century ago are

omitted from use by those upon whom responsibility for their use rests; and when a human life is sacrificed through such failure and neglect, the courts have considered such omission as constituting culpable negligence and have held those responsible therefor upon a charge of manslaughter in the second degree."

These two papers stand almost alone in the enormous recent literature which deals with Cæsarean section, for they consider the post-mortem variety of the sectio. Numerous articles look at almost every other aspect of the operation in detail; thus, in one number of the *Boston Medical and Surgical Journal* (that for 30th March 1916) there were three contributions, one on the indications, time of operating, and technique, by Dr. C. M. Green; one on modern indications for the operation, by Dr. John T. Williams; and a third entitled "Cæsarean Section—Overdone," by Dr. Foster S. Kellogg. Yet in none of these articles was the question of the post-mortem Cæsarean touched upon. Nevertheless it is of pressing importance that every medical man, and not simply the specialist in obstetrics, should be prepared to extract the child by Cæsarean section after the death of the mother. The legal aspects cannot safely be regarded as identical in America (the papers quoted are from the American standpoint) and in Britain; but too much weight must not be laid upon this difference, for till the close of the eighteenth century the law was the same in America as in this country, and further, the principles underlying any decision in such a matter may be looked on as practically similar. At any rate, Meymott Tidy in his *Legal Medicine* (1883, ii. 306) defined the position from the English standpoint in a way not fundamentally different from that occupied by Dr. Harrar. Meymott Tidy writes:—"Certain it is, that in cases of sudden death occurring in pregnant women, the surgeon should attempt the immediate extraction of the child. Although he cannot force the relatives to permit it (and not to permit it is not a punishable offence), still to refuse is as much a moral murder as the crime of producing abortion in a healthy woman." If one reads between the lines, therefore, it appears that Meymott Tidy counsels delivery without the consent of the relatives if that have been asked for and have been refused. Whitehead raises a new point, and suggests an additional responsibility when he discusses the blame which may fall on the medical man who does not deliver the child of a recently dead woman; apparently he renders himself liable to prosecution and may be found to have committed manslaughter in the second degree and be liable to its penalty.

It is quite obvious that the death of a pregnant patient undelivered places her medical attendant in a delicate position, and this may easily become one of acute embarrassment if her relatives refuse permission for the section, which may be the only means of delivering the child within the six or seven minutes which is all the time that can be safely

allowed to elapse after the maternal decease, if the infant is to have a fair chance of recovery. On the one side he may be faced with such a peremptory refusal of permission, and on the other side he is responsible for the life of the unborn child; further, he has no time for consultation and argument. The situation is certain to be a delicate one, but apparently he not only may, but he ought to extract the child on his own, and, it may be, on his sole responsibility, and, so far as statistics can give a guide, he ought to employ the Cæsarean section as the method of extraction. A further difficulty is that the death is usually sudden and may be also quite unexpected, and an added responsibility is now to be discovered in the possibility of an action for culpable negligence if surgical measures to deliver the child are not taken. Certainly no practitioner of medicine can afford to leave his conduct in such circumstances to be settled by resolutions formed for the first time in the presence of the emergency. If it were possible, the doctor's difficulties are added to when he is dealing with not a dead but a moribund pregnant woman. J. W. B.

DISEASES OF CHILDREN.

UNDER THE CHARGE OF

W. B. DRUMMOND, F.R.C.P.

Chloroma.

Gould and Le Wald reported two cases to the New York Academy of Medicine (*Pediatrics*, May 1916, p. 223). They stated that an analysis of the cases reported seemed to show that chloroma occurred more frequently in children than in adults, and more frequently in males than in females. The chief clinical features which characterised the disease were exophthalmos, which was produced by a retrobulbar lymphoma; swelling of the temporal and occipital regions, due to the involvement of the cranial bones; enlargement of the spleen and lymphatic glands throughout the body, but especially in the cervical region, and a severe and progressive anæmia causing weakness, fever, and emaciation. Hyperplasia of the bones of the skull undoubtedly began in the marrow of the cranial bones but soon involved the periosteum and the dura mater, forced the sutures apart, filled up various foramina of the skull, and by pressure upon the nerves which emerged from the brain through these openings might cause blindness, deafness, pain, or other symptoms referable to the nerves involved. While exophthalmos was the first symptom noted in a majority of the cases reported, in some cases there had been no exophthalmos. In the various reports nearly all the bones of the body had been cited as the seat of this change. The blood picture in chloroma varied greatly

in the cases reported. The anæmia was believed to be due to the rapid proliferation in the bone-marrow of the leucoblastic tissues, resulting in the crowding out of the elements from which the red blood-cells were derived. The anæmia, therefore, depended upon the deficient production of red cells rather than upon any toxic destruction of them. The hyperplasia of the leucoblastic elements led to the frequent appearance in the blood-stream of large numbers of white blood cells of various types, and had led to the classification of this condition as a form of leukæmia. In most of the reported cases this characteristic leukæmic picture had been found at some time in the course of the disease, especially during the later stages. The predominating type might be any one of the normal or leukæmic types of the leucocytes, or the hyperplasia of the parent cells might reach such a stage that the new forms did not reach the blood stream in such numbers as to give the picture of a leukæmia. During the time that the present cases were under observation the blood picture was never that of leukæmia, but this time represented but a small part of the duration of the disease. The name "chloroma" was given to the disease because of the peculiar green colour frequently seen in the tumours removed from the body, and which might also be found in any of the new growths, or even in a blood-clot within the heart, though it was a more common characteristic of the hyperplasias of the bone-marrow. Recent observations in regard to the histological characteristics of the disease showed that it did not belong to the lymphomata, carcinomata, or lymphosarcomata. Klein and Steinhaus perhaps first observed that the tumours of chloroma were largely composed of cells of leucoblastic origin. They found that neutrophile cells, especially myelocytes with eosinophile granules, were frequently predominant, and they therefore classified the disease with the leukæmias. Warthin showed that in his cases at least the tumours which involved the vertebræ and prevertebral tissues were primary in the bone-marrow spaces, that they infiltrated or grew through the lymphatics and blood vessels or broke through the bone, and secondarily involved the periosteum and surrounding tissues. Section showed that the normal marrow tissue was almost entirely replaced by cells of the large lymphocyte type, most of them showing eosinophile granulation. Transition forms of various kinds were numerous, but the whole vertebral tumour was made up of cells of leucoblastic origin with a scanty and delicate tissue reticulum, and with little evidence of new blood vessel formation. Nodules of similar histological structure were found in various organs of the body, arising from intravascular proliferation and seeming to have little influence on surrounding tissues excepting by pressure and atrophy. There was little manifestation on the part of the organ involved of a reaction in the nature of a resisting proliferation of the tissue-cells or the development of a reticulum and blood-vessels. Of two cases coming under

their observation the writers stated that the first was a female child 3½ years of age and the second was a male child 5 years of age. In neither instance was an autopsy obtained, but the Rontgen ray findings were fairly characteristic, especially in regard to the proliferation of bone tissue in the skull producing a separation of the suture lines. In the second case there were characteristic changes in the long bones. The process seemed to be essentially a rarefaction and proliferation in the medullary cavities combined with a subperiosteal change lifting the periosteum over a variable distance from the surface of the shaft. The condition was distinguished from infantile scorbutus and rachitis by the absence of epiphyseal changes; from a myelomatous condition by the combination of the periosteal changes with the changes in the medullary cavity, and from sarcoma by the extent of the process. The changes in the medullary portion of the bone appeared to be distinctly different from those of syphilis. The gross pathological changes in both of these cases were in exact accord with those described by Warthin. As far as the writers had been able to ascertain, these were the first cases of chloroma in which the Rontgen ray findings had been recorded.

The radiograph of the first patient, taken 29th January 1913, showed marked enlargement of the mediastinal lymph nodes with slight displacement of the trachea to the right. Another radiograph taken three days later confirmed the finding of enlargement of the mediastinal lymph nodes. Examination of the skull on 5th February showed a distinct separation of the coronal sutures producing a wedge shaped gap measuring 1 cm. at the vault. Radiographic exhibitions of the head on 12th February confirmed the previous finding of separation of the coronal sutures, and also showed a separation of the parietal sutures. Stereoscopic examination confirmed the findings of separation of the sutures of the skull, including those of the occipito parietal regions. The first radiographic examination of the second patient, made 22nd October 1913, showed a distinct separation of the suture lines, especially marked in the region of the coronal and the parietal sutures. The separation of the frontal sutures measured about 2 cm. at the vertex. Examination of the chest showed an extensive involvement of the mediastinal lymph nodes. The diaphragm was pushed upwards on both sides, indicating a distinct enlargement of the liver and spleen. The bones of the upper extremities showed marked changes of a very peculiar type. The medullary portion of the long bones showed a peculiar mottling due to the rarefied areas. The periosteum was distinctly raised from the surface of the bone over the greater portion of the shaft. The peculiar combined involvement of the medullary cavity and the periosteum had not been observed in other bone lesions; from these findings a diagnosis of chloroma seemed justified, when taken in conjunction with the history of the case. Radiological

examination of the lower extremities showed an involvement of the long bones exactly like that observed in the upper extremities.

Infant Feeding.

Vining (*Practitioner*, Nov. 1915) protests against the almost universal use by the poorer classes of diluted milk in the feeding of infants, and pleads for the more extended use of whole citrated milk. He suggests that the extensive use of diluted milk may have something to do with the present appalling rate of infantile mortality and the distressing amount of general ill-health among the children of the poor. Pritchard (*Amer. Med.*, 1916, xi.) admits that whole-milk feeding usually gives good results, but considers that it does not give the best results. Instead of modifying the specific physiological make-up of a child to a certain kind of food, a task which may be impossible, it is better to adapt the food to the physiological idiosyncrasies of each case. The dilution of infants' foods is too often excessive, the success of the "whole-milk" method being probably due to the limitation in the amount of water. Individual adaptation is essential to the successful administration of foods to infants. If the principles of percentage feeding are understood, synthetic foods can be prepared in many ways to satisfy physiological requirements. Dried milk, if properly modified, has all the advantages and few of the disadvantages of dairy milk.

Vining (*Pediatrics*, May 1916) considers that the plan of prescribing an individual dietary for every infant born is "neither practical nor possible." The majority of infants can never be under constant medical supervision, and there is therefore a danger in making infant feeding too complicated or too specialised.

Zimmermann (*Pediatrics*, May 1916) discusses disturbances in health due to "Some Common Errors in Infant Feeding." Such disturbances are usually due (1) to too little food. In such cases the infants sleep well, but do not gain weight. The stools are few and scanty, and the urine also is scanty. The temperature is usually subnormal, except in the early days of life, when there may be marked fever. (2) Over-feeding leads to dyspepsia, colic, diarrhœa, and vomiting, or the regurgitation of small quantities of food. The stools are often green, and contain mucus and curd. When long continued, facial eczema is frequent. (3) Excess of fat does not necessarily mean that the amount of fat is large, but that it is above the tolerance of the infant for milk fat. The children are fretful, pasty in appearance, and alternately lose and gain in weight. The stools are characteristic, being white or grey in colour, hard, dry, and of a foul odour. Calcium and magnesium are excreted in large quantities. Fat and calcium must be present in the food in large quantities before soap stools can result. (4) Protein is rarely to blame for disturbances. We cannot prove that the protein of cow's milk, in quantities two or three times that

of breast milk, is injurious to the infant. (5) Excess of carbohydrate causes diarrhœa, vomiting, and slight fever. Infants under three months are most likely to suffer. The most common faults are overfeeding, irregular feeding, and too frequent feeding. Whether infants are fed at the breast or artificially, the four hour interval gives excellent results. W. B. D.

PATHOLOGY.

UNDER THE CHARGE OF

THEODORE SHENNAN, M.D., AND JAMES MILLER, M.D.

Sporotrichosis of Bone.

The author, D'Agata (*L' Policlinico*, 15th January 1916, p. 1), refers to 15 reported cases of sporotrichosis of bone in which the bones affected were mostly the tibia, and in single cases the bones of the forearm, the humerus, metatarsal, calcaneum, metacarpal, sternum, and bones of the orbit.

The lesion usually gives vent to a viscid, purulent, brownish-yellow discharge, and shows microscopically a granulomatous structure suggesting tuberculosis or syphilis. There is frequently a history of previous slight injury of the part affected. In the author's case the superior maxilla was affected. The pus contained great numbers of polymorphonuclears and some mononuclears; many elements like pyknotic detritus of nuclei of leucocytes; short diplobacilli and spirochætes (sp. refringens and dentium); actinomycosis was excluded microscopically. Wassermann's reaction proved negative. On Sabouraud's acid glucose medium, at room temperature, a growth of sporotrichum Beurmanni developed slowly. Intraperitoneal injection of 0·5 c.cm. of a broth culture produced in the rat a peri-orchi-epididymitis. Positive agglutination with the cultures confirmed the sporotrichosic nature of the disease. In his case the tibia was also affected, but no history of injury or irritation of the parts could be obtained.

On reviewing the reported cases one concludes that infection had taken place either through the skin by sporothrix-carrying material (thorn, wood-splinter, bark, etc.), or that an injury had caused localisation of the infective agent circulating in the blood. In the latter event the primary invasion had probably occurred through the bucco-pharynx, where the sporothrix may live saprophytically, or from the intestine, to which the organisms may be introduced in raw or insufficiently cooked material.

Most of the cases had been engaged in work implying handling of dry vegetables, straw, bark, or wood—all sporothrix-carrying material.

In the bones the lesions resemble more or less closely the subacute or chronic osteomyelitis of tuberculosis and syphilis, and like these, may

either resolve, or soften and become purulent, or undergo sclerosis. Even when lesions are undergoing fibrosis or sclerosis, suppurative foci can be found centrally. This is a point of value in distinguishing these cases from osteomyelitis fibrosa or cystica, which may occur in similar positions. The lesions as a rule attack the medulla and compact bone, and only invade the periosteum secondarily; occasionally the periostitis is primary.

It is not always possible to demonstrate the parasites in the local lesions. De Beurmann, Gougerot, and several others state that in the human lesion the parasite is short, ovoid, 2 to 10 μ in length by 1 to 3 μ in breadth, with finely granular protoplasm, less deeply stained at the ends, and surrounded by a thin colourless membrane. They occur lying free or lying within small or large phagocytes.

It can be cultivated upon Sabouraud's glucose agar, at the room temperature.

For details as to the preparation of Sabouraud's medium the reader is referred to *Les nouvelles mycoses*, De Beurmann et Gougerot, p. 121.

Intraperitoneal injection of a male rat or mouse after a variable time (10 to 20 days) produces a characteristic swelling of the scrotum with orchitis, in the lesions of which the parasite is very abundant.

To confirm or to make the diagnosis, the sporo-agglutination reaction of Widal and Abrami may be employed, the serum of patients agglutinating a homogeneous emulsion of sporotrichum Beurmanni in dilutions of $\frac{1}{1500}$ to $\frac{1}{600}$, whereas actinomyces, discomyces, oidium are agglutinated in dilutions of $\frac{1}{50}$ to $\frac{1}{150}$. Additional diagnostic methods available are the fixation of complement (Widal and Abrami): the meiostagmin reaction (D'Agata): the cuti-reaction (De Beurmann and Gougerot); the opsonic index (Milhit); the precipitin reaction (Widal, Sicard, and Gougerot); but these all require special technique and very considerable laboratory experience.

As already remarked, one of the interesting points in connection with sporotrichosis ossea is the fact that it frequently attacks bones, and parts of these in which fibro-cystic osteomyelitis occurs. Another point is the importance of keeping in view the possibility of a sporotrichosic causation in all cases of subacute or chronic osteomyelitis presenting unusual features.

Etiology, Mode of Infection, and Specific Therapy of Weil's Disease (Spirochetosis Ictero-Hemorrhagica).

The authors, Inada, Ido, Hoki, Kaneko, Ito (*Journ. Exp. Med.*, March 1916, p. 377), believe that an epidemic and endemic disease occurring in several districts of Japan corresponds closely with, if indeed it is not the same as, Weil's disease, which occurs endemically in Europe and America. The disease is characterised by conjunctival congestion, muscular pains, fever, jaundice, hæmorrhagic diathesis, and albuminuria.

The mortality of the Japanese disease is 32 per cent., and splenic enlargement is rare—about 10 per cent.

In beginning their investigation of the disease the authors failed to discover bacteria in the blood, urine, and fæces. Then on inoculating monkeys, rabbits, rats, and guinea-pigs with the blood of patients, they found that guinea-pigs developed albuminuria, conjunctival congestion, jaundice and hæmorrhage when the inoculated blood was taken during the first 7 days of the disease. Spirochætes were found in great numbers in the livers of the guinea-pigs; in smaller numbers in the blood. In patients dying on the 6th day numerous spirochætes are found in the liver.

Out of 17 guinea-pigs injected intraperitoneally with the blood of patients in the 4th to 7th day of the disease, 13 gave positive results, jaundice appearing in 7 to 8 days, and death taking place 24 hours thereafter. Rabbits proved insusceptible, and white rats were less susceptible than guinea-pigs, which appeared to be more susceptible than man.

The causal organism, which they have named the spirochæta icterohæmorrhagica, varies in length from 6 to 9 μ to 12 to 13 μ. The extremities are sharp and usually hooked. The undulations are not so regular as those of the treponema pallidum, there being usually 2 or 3 large irregular waves, and 4 to 5 smaller waves.

To demonstrate it, films from blood or liver are fixed in absolute alcohol, methyl alcohol, or osmic acid; and stained in Giemsa's solution (3 drops to 2 c.cm. of water). This is applied for at least 2 hours, until the granules of the leucocytes are deeply stained. The spirochætes stain red or purplish-red. For vital staining borax methylene blue may be used. With dark ground illumination the organism is easily seen. It is irregularly refractile, dark and light parts alternating. It divides probably by transverse fission. It has been cultivated according to Noguchi's methods, portions of guinea-pig's kidney being added to the ascitic fluid in place of the rabbits' testes used for the treponema pallidum. The fluid does not coagulate, and no turbidity develops.

Anaerobic conditions are assured by a layer of liquid paraffin on the top of the culture medium.

The optimum temperature is 22-25° C.

The organism probably enters by the alimentary tract, but this has not been finally determined experimentally; it can penetrate either through the skin or the alimentary tract.

It is eliminated probably chiefly by the urine. Inoculation of urine, fæces, and bile has caused the disease in guinea-pigs. Experimental animals contain it in the urine and fæces.

On examining infected guinea-pigs after death there is marked jaundice, with hæmorrhage in different tissues and organs, especially in the lungs. The liver-cells are cloudy; there is acute parenchymatous nephritis; the spleen is enlarged and congested. Spirochætes are found

mostly in blood, liver, suprarenals and kidneys, between the cells or within phagocytes.

In man after death spirochætes are found most readily in the kidneys, fewer in the liver, lymph glands and spleen, cardiac muscle, voluntary muscle, testes, and walls of blood-vessels. At times they can be found in pancreas, intestinal wall, gall-bladder, genitals, central nervous system, but in these parts they are usually degenerating.

Immune substances can be recognised by Pfeiffer's phenomenon in the body of infected individuals from the 14th to 15th day of the disease, and they persist for years. An attempt has been made to immunise guinea-pigs by injecting repeatedly emulsion of infected liver, and finally by injecting pure cultures of the spirochæte killed by the addition of carbolic acid.

For prevention of infection it is of especial importance to disinfect urine of patients for at least 40 days from the beginning of the illness, that being the period during which spirochætes persist in the urine. The faeces and bloody sputum should also be disinfected.

NEW BOOKS.

The Involuntary Nervous System. By WALTER HOLBROOK GASKELL, M.A., M.D., F.R.S. Monographs on Physiology, Edited by ERNEST H. STARLING, M.D., D.Sc., F.R.S. Pp. ix. + 186. With Coloured Figures. London: Longmans, Green & Co. 1916. Price 6s. net.

THIS is the first of a series of monographs which are to be published under the editorship of Prof. Starling. Each monograph will contain an account of our knowledge of some particular branch of physiology, written by someone who has contributed to the attainment of our present position. "An exhaustive account of previous writings is not aimed at, but rather an appreciation of what is worth retaining in past work, so far as this is suggestive of the paths along which future research may be fruitful of results." The MS. of the present volume was only completed by Dr. W. H. Gaskell a very short time before his death.

The first chapter discusses the history of the involuntary nervous system. "Everything points to the conclusion that the so-called sympathetic nervous system consists simply of masses of motor neurons to a special system of muscles, which have left the central nervous system, but are still connected with it by connector fibres in the rami communicantes." There are three main "outflows" of connector fibres —the mid-brain, the thoracico-lumbar and the bulbo-sacral. Of these, the thoracico-lumbar or sympathetic outflow only connects with all the

excitor neurones of the sympathetic system. The characteristic motor functions of the nerve cells belonging to these three outflows are considered in great detail. The problem of the inhibitory nerves is next considered, also at considerable length. "In the case of the blood-vessels of all glandular tissues it cannot be said that the separate existence of vaso-dilator nerves is proved without doubt." The whole question of vaso-dilator nerves and their relation to the sensory nerves of the skin is one which is only beginning to be understood. "Perhaps the safest way of looking at the matter in our present state of knowledge is to hold that vascular dilatation of an organ can take place in two ways, either by alterations in the chemical constitution of the fluids bathing the muscles of the small arteries, or by the stimulation of nerve fibres which relax those muscles." The rhythmic and peristaltic movements in the involuntary muscles of the vertebrate are discussed in a separate chapter, and it is concluded that although muscle has the power of contracting rhythmically it is dependent on its innervation for its regulation. The innervation of glandular structures is next considered. It is specially to be noted that there is no evidence that the kidneys are under the control of the nervous system at all. The kidney cells secrete substances brought to them by the blood, and the activity of these cells is determined by the chemical nature of these substances.

A very interesting chapter is devoted to the phylogenetic origin of the sympathetic nervous system and its close relationship to the chromaffin tissue system. "The evidence of the lowest vertebrate points to the origin of the sympathetic nervous system from nerve cells in the central nervous system of some invertebrate, which are motor to the vascular system and contain adrenaline in their substance."

The book closes with a summary of the various systems of involuntary muscles which occur in vertebrates, and the various nervous groups or systems which are connected with them, their inter-relationships and possible evolution. There is a bibliography.

This is a most stimulating work, requiring careful and prolonged study.

Treatise on Fracture. By JOHN B. ROBERTS, A.M., M.D., F.R.C.S., and JAMES A. KELLY, A.M., M.D. Pp. xxv.+677. With 109 Illustrations. Philadelphia and London: J. B. Lippincott Co. 1916. Price 25s. net.

THIS book, according to the preface, is "to supply the student and medical practitioner with a clear, concise, and systematic presentation of the subject of fractures."

The first chapter is devoted to general considerations, classifications, complications, and various methods of treatment. Strangely enough,

for a book intended for the use of students and medical practitioners, the second chapter deals with the operative treatment of closed fractures, with numerous illustrations. Fortunately, stress is laid on the need of a skilled surgeon to carry out such procedures, and quotations are made from the American Surgical Association's Fracture Committee to this effect. The rest of the book is devoted to individual fractures and their treatment. Ignorance of the reader is implied by the attention to ordinary anatomy, and an expert knowledge by the casual mention of various splints, etc., without those minute details which are necessary for a student or medical practitioner. The illustrations and radiograms are good, especially the methods of indicating the anæsthesia associated with various spinal lesions. Much stress is thrown on the somewhat doubtful influence of muscles as a cause of displacement when long bones are fractured.

For its size the book is not well adapted for the needs of a student or medical practitioner.

Notes on the Causation of Cancer. By The Hon. Rollo Russell, With a Preface by Dr. Dawtrey Drewitt. Pp. xii. + 116. London: Longmans, Green & Co. 1916. Price 3s. 6d. net.

The author of this book, the Hon. Rollo Russell, who had written two previous books on the subject of malignant disease, unfortunately, after months of careful and patient labour on the work under notice, died before its publication.

It has been said that "Cancer is the casket of its own secret": and a book dealing with its causation can only deal with hypotheses and not with certainties.

As an Oxford graduate who had studied chemistry under Sir Benjamin Brodie and Harcourt, Russell attained the delicate precision required in chemical experiments, and all his life had a strong scientific bent.

The book is devoted to statistics, and to an attempt to discover what elements, if any, tend to produce a malignant condition. One finishes it, however, feeling somehow that one is not any the wiser.

The summary of conclusions is practically this, that meat, tea, and alcohol are mainly responsible. In his own words: "On every side we are invited to consume deadly food, deadly drink, deadly drugs, and to try vainly to cure ourselves without changing our self-destructive habits by correcting loud-voiced parasites crying seductively to us to comfort or enjoy ourselves with beverages which science well knows are the capital sources of crime, disease, and death." None of the three causes, however, seem entirely satisfactory in view of other considerations. The eating of flesh cannot be entirely responsible, since cancer is very prevalent among the Hindoos, who do not eat meat.

That it is not due to alcohol is abundantly proved by the fact that more women die from malignant disease than men, and women, as a class, are more temperate, while many sufferers from cancer have been life abstainers. Neither can tea and coffee be held exclusively responsible, looking to the fact that women, who suffer from cancer more than men, and are much greater consumers of tea, have a much lower death-rate from carcinoma of the stomach and œsophagus.

The book is a very readable one to all who are interested in the subject, although we do not agree with its penultimate statement that our Dreadnoughts grow while our men decay. The answer is to be found on the battlefields of Northern France.

The Mortality from Cancer throughout the World. By Frederick L. Hoffmann, LL.D., F.S.S., F.A.S.A. Pp. xvi. + 826. Newark, N.J. : The Prudential Press. 1915.

This is truly a remarkable book, and one which is invaluable to all students of the cancer problem. It extends to nearly 900 pages, over 200 of which are devoted to a most interesting summary of the charts and tables of statistics which form the bulk of the volume.

Among the subjects dealt with in the text are the statistical basis of cancer research, the increase of cancer which figures absolutely prove, the occupational mortality, and the geographical incidence throughout the world. Naturally the American states and cities have been most thoroughly detailed.

Perhaps the most interesting chapter, however, to the surgeon and general practitioner alike, is the concluding one dealing with some general observations and conclusions on the cancer problem.

It is the best *resumé* of the present knowledge on the subject which we have yet seen, and summarises not only the statistical position, the supposed causes and affinities of malignancy, the pros and cons of the parasitic theory and other matters, but even summarises the recent technical literature on the subject.

The charts and tables have been prepared with great care, and, carefully studied, reveal many strange facts.

Mr. Hoffman has proved what Roger Williams suggested some years ago, that cancer increases in frequency with distance from the equator, as is shown by his table on page 142.

This table is very striking. It shows that in the great cities of the world between latitudes 60° N. and 50° N. the cancer death-rate per 100,000 living is 105·7 ; in the cities between 50° N. and 40° N. it drops to 92·4 ; between 40° N. and 30° N. it is 78·1 ; while between 30° N. and 10° N. it actually drops to 42·3.

Incidentally it may be pointed out as a curious fact that when

Heligoland was British its cancer mortality-figure per 100,000 living was in 1869-70 5·4 ; in 1903 it had risen to 140·3 (Table 104, Appendix G).

Mr. Hoffman's statistics also show that the American negro has actually as high a death-rate now as the white man, although his comparative mortality some years ago was very much less, the increase being from 39·1 to 73·5 in the last 20 years ; that the condition is extremely rare among the North American Indians ; that the supposed immunity of the Jew is a myth ; that cancer of the female generative organs in England and Japan is not statistically far apart, whereas cancer of the breast is ten times as common in England as it is in Japan ; that the cancer death-rate in Denmark and the Danish West Indies, which were sold the other day to America, are both very high, while the surrounding districts in the west have a very low mortality, and many other strongly suggestive facts. Among other details Mr. Hoffman points out that cancer now in the civilised countries of the world alone results in the appalling annual loss of at least 500,000 lives.

As a work of reference this book on the mortality of cancer throughout the world is a classic.

Links in a Chain of Research in Syphilis. By J. E. R. M'Donagh, F.R.C.S. Pp. 199. London : Harrison & Sons. 1916.

This book is Mr. M'Donagh's Hunterian Lectures of 1916 in printed form. It may be said to consist mainly of a plea for the author's method of treating syphilis by the use of alternating injections of reducing agents such as sulphur ("Intramine"), and of oxidising agents such as iron ("Ferrivine").

In support of this treatment, chapters are devoted to the bacteriology of syphilis, to the rationale of dyeing and staining, to the Wassermann reaction, to immunity, to anaphylaxis, and, above all, to chemotherapy. The latter subject occupies over sixty pages, and is of such a highly technical character that really only experts in this particular department of medicine are competent to criticise it. The fact that the author's views are admittedly heterodox on most subjects connected with syphilis adds to rather than detracts from the interest of an exceedingly clever and well-written book. As regards the Wassermann test, Mr. M'Donagh considers that it is not in any way a specific reaction, and that a positive result does not necessarily signify that the patient has active syphilis, or that he requires further treatment. Equally dogmatic are his references to "606," as will be seen from the following (page 120): "*Fortunately*"—the italics are our own—"there is hardly a statement of Ehrlich's which now holds good, and I am of the opinion that, before very long, not one of the

arsenical compounds will remain in general use." In 1912 (*Salvarsan in Syphilis*, page 149), Mr. M'Donagh wrote—"It would not be possible for any writer who accepts as proven the verdict that salvarsan cures syphilis, to lay down his pen before it had rendered honour to whom honour is most justly due. Ehrlich has done mankind a service which takes rank along with those of Lister and Jenner. To him the thanks will come—as it has always come to the truly great—when his fellow men are worthy to understand him: which generally happens in the succeeding century." One wonders how far the war with Germany has influenced this striking change in the author's views.

Laboratory Manual in General Microbiology. Prepared by the LABORATORY OF BACTERIOLOGY, HYGIENE, AND PATHOLOGY, OF MICHIGAN AGRICULTURAL COLLEGE. Pp. 418. London: Chapman & Hall. 1916. Price 10s. 6d.

THIS laboratory manual serves admirably the needs of the student in introducing him to the study of micro-organisms. It is written primarily to give a working knowledge of laboratory methods to students of agriculture and sanitation, but equally well meets the needs of medical students. The value of the book lies in the inductive method used, so that the student is stimulated to think for himself, and independently arrives at his conclusions from the evidence of the experiments performed. The exercises are clearly and concisely written, and so arranged as to be useful as a reference book for laboratory methods. The author has succeeded in writing a manual which serves as a guide to the student but not as a prop on which to lean. On this score, as well as on the accuracy and simplicity of its exercises, this book can be recommended to teachers as a laboratory manual for beginners in microbiology.

I. K. Therapy in Pulmonary Tuberculosis. By WILLIAM BARR, M.D., D.Sc.(Glas.), D.P.H.(Camb.). Pp. 42. With 42 Charts. Bristol: John Wright & Sons. 1916. Price 3s. 6d. net.

THIS book sets forth the writer's experience of I. K. therapy, and we frankly confess that we lay it down with a feeling of disappointment. After reading in the eighteen lines which describe the action of I. K. that "the lysins destroy the bacteria causing tuberculosis, and the toxins liberated, as a result of this lysis, become neutralised by the antitoxins," we naturally expect great results which are not forthcoming. Any agent which can destroy the bacilli *in situ* and neutralise the toxins liberated has a great future before it, but we do not feel con-

vinced, on the evidence before us, that I. K. has established the claim to work such wonders.

A study of the summary of cases shown in the book tends to confirm this, as there is nothing to show that any remedy of exceptional potency has been administered. The writer truthfully admits that it is extremely difficult to give statistics of results, as "one must recollect the great tendency of the disease to arrest itself spontaneously." He apparently, however, places considerable weight as to the value of treatment on the statements of the patients, as after all, he says, "It is the patient who, is the final judge." In the next sentence we have: "It is, however, on clinical evidence that any treatment must stand or fall," and then follows the summary of a series of 47 cases already referred to.

To one beginning the treatment of pulmonary tuberculosis with I. K. the perusal of the book would be interesting and helpful.

NEW EDITIONS.

Manual of Operative Surgery. By JOHN FAIRBAIRN BINNIE, Kansas City. Seventh Edition. Pp. xv. + 1363. With 1597 Illustrations. London: H. K. Lewis & Co. 1916. Price 32s. net.

IT is little more than two years since we recorded the appearance of the sixth edition of this manual of operative surgery, and already a new edition is upon us. The author states that "in spite of much new material careful pruning has prevented any great increase in the size of the volume." In our last notice we ventured to suggest that a certain amount of pruning would be of benefit, but we can scarcely commend that form of pruning which has added 112 pages of subject-matter. It is true that an "Appendix on War Surgery" accounts for about 30 of the additional pages, and that the number of illustrations has been increased by about 160, but even allowing for these additions there is still a considerable increase in the text. Much of the work that has been published since the last edition appeared has been incorporated in this edition. The surgery of the blood vessels is very fully dealt with, and a section has been included on cardiac surgery "even although, up to the present, such work has been mostly confined to the physiological laboratory."

The appendix on war surgery has been contributed by Dr. Walker S. Sutton, who has summarised the more accessible war literature in a somewhat sketchy manner. The effects of the different antiseptic agents that have passed under review since the war began are quoted by the

writer, but there is nothing to show that he writes from personal experience of their action. "Excision and primary suture" (of gunshot wounds), he says, "has been carried out by Gray with surprising success." We share in his surprise, but then it may be that the cases in which we have seen the results on this side have not been "properly selected," which is one of the qualifying postulates for the success of the procedure. A selection of illustrations of the numerous splints devised to obtain extension for cases of compound fracture of the extremities is given, and detailed instructions for the use of Steinmann's pins. Considerable space is devoted to the important subject of the localisation of foreign bodies. There is no consideration of regional surgery. On the whole, this section merely serves to give the work an up-to-date appearance without adding appreciably to its value.

For the rest, we still consider this one of the best works of reference on operative surgery for the advanced student or the operating surgeon.

The Diseases of Women. By Sir JOHN BLAND SUTTON, F.R.C.S.(Eng.), LL.D., Surgeon to the Middlesex Hospital, and ARTHUR E. GILES, M.D., B.Sc.(Lond.), F.R.C.S.(Edin.), Senior Surgeon to the Chelsea Hospital for Women, and Gynecologist to the Tottenham Hospital. Seventh Edition. Pp. xv. + 571. With 150 Illustrations. London: William Heinemann. 1916. Price 15s. net.

IT is close upon twenty years since Bland Sutton and Giles' *Diseases of Women* first made its appearance, and during all these years it has held a foremost place amongst gynecological text-books for students and practitioners. In its seventh edition it appears in a new garb of sombre hue, which is by no means so attractive as the colours we remember it to have flaunted in its earlier days. This, however, is the only adverse criticism we have to offer, and, we may add, the only sign which it shows of advancing years!

We congratulate the authors upon having adopted the pathological system of classification of the various diseases in place of the older-fashioned anatomical arrangement. In regard to the all-pervading subject of pelvic inflammation the latter system was always calculated to give the student a fragmentary and unscientific impression, and has wisely been discarded in most modern text-books.

The whole subject of treatment has been taken up in one special section, a plan which in a book of this size makes for greater conciseness. There is also a valuable section on prognosis which will be of great help to the practitioner.

Several new illustrations have been added, and in all respects the high standard of the earlier editions has been more than maintained.

Aids to Obstetrics. By Samuel Nall, B.A., M.B.(Cantab.), M.R.C.P. (Lond.). Revised by C. J. Nepean Longridge, M.D.(Viet.), F.R.C.S.(Eng.), M.R.C.P.(Lond.). Eighth Edition. Pp. viii. + 216. London: Baillière, Tindall & Cox. 1916. Price 2s. net.

For its size and for the purposes at which it is aimed the *Aids to Obstetrics*, originally written by Dr. Samuel Nall, and brought up to date by Dr. Nepean Longridge, is a most admirable volume. It makes no pretence at being a text-book, but is designed to help students preparing for examinations to revise the subject rapidly. We feel sure that the pious hope expressed by the reviser, that it may also prove of practical value to the practitioner, will also be realised, for the information contained in it is thoroughly accurate, and the teaching in accord with the most recent views. The fact that it has run to its eighth edition is the best proof that it has met a real want.

Anatomy, Descriptive and Applied. By Henry Gray, F.R.S. Nineteenth Edition. Edited by Robert Howden, M.A., D.Sc., M.B., C.M. Pp. 1304. With 1143 Illustrations. London: Longmans, Green & Co. 1916. Price 32s. net.

It is a significant fact that this volume, in the face of numerous competitors, still maintains its position in the front rank of text-books of anatomy. Published first in 1858, it has now reached its nineteenth edition, and its popularity among students and teachers of anatomy remains undiminished. The present edition has been brought up to dated by the introduction of the Basle nomenclature throughout. It would appear that the new terminology has come to stay in spite of its Teutonic origin, and it has undoubtedly justified its introduction, as is readily appreciated from the greater facility with which the present student of anatomy understands and remembers anatomical terms.

The secret of the success of *Gray's Anatomy* lies very largely in the methodical and orderly way in which the book is written. It gives a very complete account of the whole of human anatomy and at the same time avoids mention of debatable matter or of questions which are unnecessarily advanced for the ordinary student of anatomy. This feature is especially evident in the section on embryology: this subject is laid down in an admirably lucid and dogmatic manner without encroachment on questions which are not yet settled or which are unnecessary to a clear understanding of development.

It may be questioned as to whether the applied anatomy which is included in the volume is of any great value, scattered as it is throughout the whole book. The ordinary student of anatomy is unable to appreciate the pathological descriptions which are given,

while the senior student will consult a work on surgical anatomy rather than waste his time hunting up these isolated paragraphs.

The illustrations are the least satisfactory feature of the book; in most sections they are considerably below the standard which is expected in a modern text book of anatomy. It is especially difficult to understand how the diagrams of fractures which illustrate certain of the paragraphs of applied anatomy have been retained in the present edition: in view of the results of X ray examination these diagrams are far from giving correct examples of the ordinary types of the fractures indicated. In the section on surface anatomy a subject of better muscular development and less adiposity might have been obtained for the illustrations.

Apart from these minor defects there is nothing to criticise. Professor Howden is to be congratulated on the way in which the text has been kept in line with recent changes. The present edition is certain to maintain the popularity and success of this work.

NOTES ON BOOKS.

Reports, Transactions, etc.—*The Johns Hopkins Hospital Reports*, Vol. XVII. (Baltimore: The Johns Hopkins Press, 1916.) Vol. XVII. of the Johns Hopkins Hospital Reports, which has just been issued, contains eight papers. Two of these papers deal with the formation of thrombi in a failing circulation. Hewitt discusses "Free Thrombi and Ball Thrombi in the Heart," a condition which, as he points out, was first described by Dr. Wood in 1814 in the *Edinburgh Medical and Surgical Journal*. These rare thrombi are found in the left auricle in cases of mitral stenosis and insufficiency. It has never been known that they act as ball valves. In a paper on "Venous Thrombosis during Myocardial Insufficiency" Sladen and Winternitz adduce evidence which suggests that the formation of thrombi in this condition of the heart is related to infection.

Two papers deal with the pathology of blood diseases: the first, by Selling on "Benzol as a Leucotoxin," was first published in Ziegler's *Beiträge* in 1911. In the second, on "Leukæmia of the Fowl," Schweisser describes the blood in the fowl in a case of spontaneous leukæmia, and showed that by injecting an emulsion of the liver and spleen this disease could be transmitted to the fifth generation.

In a paper on "Primary Carcinoma of the Liver" Winternitz discusses the classification of cancers of the liver and the conditions with which they are associated. He brings out again the striking fact that the liver is rarely the seat of primary carcinoma, but that it is one of the commonest sites of secondary growth.

A paper by Miss Florence Sabin on the "Origin and Development of the Lymphatic System" forms an important contribution to a subject of fundamental interest in physiology and pathology. The general conclusion is that the lymphatic system develops from the veins and continues to grow by sprouting of the endothelium. Certain tracts of the body, of which the nervous tissues are an example, are never provided with lymphatics.

Malone describes, with ample illustrations, the structure of the cells found in the nuclei of the optic basic ganglia and its neighbourhood.

In a paper on the "Statistical Experience Data of the Johns Hopkins Hospital, 1892-1911," Dr. Hoffmann points out that the records of American hospitals have been largely neglected by the medical statistician. The same criticism would apply to the use made of hospital records in this country. The hospitals are yearly accumulating a great mass of data regarding methods of diagnosis and treatment and their relation to prognosis. "The necessary scientific classification, tabulation, and periodical publication of hospital statistics is neither a very difficult nor an expensive task." Dr. Hoffman's paper includes an elaborate analysis of the Johns Hopkins data. The extension of insurance to a national undertaking which is gradually taking place must bring it about that hospital data will be required to provide a basis of calculation, and from this point of view the paper possesses unusual value.

From these notes the reader will gather a conception of the varied interest and importance of the problems dealt with in the papers included in this volume.

The Child Welfare Annual, Vol. I. (John Bale, Sons & Danielsson, Ltd.), in the words of the editor, Dr. Kelynack, "aims at being an authoritative guide and directory to the chief agencies in the United Kingdom working for child betterment." The task is somewhat overburdened by the loose interpretation given to the term "Child Welfare," for the list includes many agencies only remotely connected with the subject as generally understood, and the basis of selection is difficult to follow. The alphabetical arrangement is confusing when applied to schemes and agencies so very diverse as those represented here. More effort at grouping the various schemes under headings would have repaid the trouble and simplified the labours of the compiler. There is ample scope for a work of this kind even if the problem had been limited strictly to the aspects which are the subject of the Local Government Board's recent recommendations. To deal effectively with more than this would demand a much larger work.

The volume opens with a review by the editor of recent developments of child welfare work, in which is incorporated in full the memoranda of the Local Government Boards of England (1914) and Scotland (1916). An interesting and suggestive survey of the subject, entitled

"The Coming Race," by Dr. F. J. Allan, Medical Officer of Health for the city of Westminster, stands out among the original articles.

The list of "schemes" is incomplete and not very representative. Out of the very large number now at work only 36 are given in this section, and more than one of this number fails to gain the attention it deserves. Of the work in Manchester, for instance, only most cursory mention is made in a general statement by the secretary of the Children's Homes and Aid Society, yet Manchester was the second city in the kingdom to start "Schools for Mothers," and has been particularly successful in developing this work on a voluntary basis until it was taken over recently by the Corporation. An account of the scheme by one of those responsible for it might well have found a place here.

The secretaries of a number of institutions furnish useful statements regarding their work. Publications bearing on child welfare are reviewed, and notes given on preparations, appliances, etc., likely to be of service in child welfare work. Some memoranda, and lists of health societies, school clinics, industrial schools, etc., close the volume, which has the promise of a future of usefulness.

True to its traditions, the *Medical Annual* for 1916 (John Wright & Sons, Ltd., price 10s. net) keeps us fully abreast of the medical literature of the last twelve months. As was to be expected, the surgery and medicine of war bulk largely, not only throughout the various sections of the work, but also in a special chapter devoted to the general questions of naval and military surgery, contributed by Deputy Surg.-Gen. A. Gascoigne Wildey, R.N., which is excellent. Another specially useful chapter on "Wound Infections" is written by Capt. A. C. Inman, R.A.M.C., which condenses the voluminous literature on this subject which the war has produced. It is enough to say that this issue maintains the high level to which the able staff of contributors have made us accustomed.

The third edition of a very useful little book for nurses and junior students—*Urine Examination Made Easy*—by Dr. Thomas Carruthers (Churchill, price 1s. net) has appeared. The directions are admirably clear, and the selection of methods is discriminating.

Another small book for nurses which we can commend is Miss Catherine W. Smart's *Bed Sores—Their Prevention and Cure* (John Bale, Sons & Danielsson, price 1s. net).

BOOKS RECEIVED.

Asshetон, R. Growth in Length . . . . . (*Cambridge University Press*) 2s. 6d.

Berry, J., F. M. D. Berry, and W. L. Blease. The Story of a Red Cross Unit in Serbia . . . . . (*J. & A. Churchill*) 6s.

Cable, F. M., and C. W. Cathcart. A Surgical Handbook. Seventeenth Edition . . . . . (*J. Wright & Sons, Ltd.*) 8s. 6d.

Calendar of the University of Toronto, Faculty of Medicine, 1916-17 . . . . . (*University Press*) —

Collected Papers of the Mayo Clinic, Rochester, Minnesota. Vol. VII. 1915 . . . . . (*W. B. Saunders & Co.*) 5 vols. 5.

Cullen, T. S. Diseases of the Umbilicus . . . . . (*W. B. Saunders & Co.*) —

Dearborn, G. van N. The Influence of Joy . . . . . (*The Boston Medical*) —

Ding wall-Fordyce, A. Diseases of Children . . . . . (*J. & A. Churchill*) 10s. 6d.

Eden, T. W., and C. Lockyer. Gynecology for Students and Practitioners . . . . . (*J. & A. Churchill*) 25s.

Fraser, H. The Bacteriology of Dysentery in Malaya . . . . . (*Studies from the Institute for Medical Research*) 3s. 6d.

Graves, W. P. Gynecology . . . . . (*W. B. Saunders & Co.*) —

Hutchison, R. Food and the Principles of Dietetics. Fourth Edition . . . . . (*Edward Arnold*) 16s.

International Clinics, 1916. Vol. II. Twenty-sixth Series . . . . . (*J. B. Lippincott Co.*)

Jellett, H. A Practice of Gynæcology . . . . . (*J. & A. Churchill*) 24s.

Krehl, L., and A. F. Briefld. The Basis of Symptoms. Third Edition . . . . . (*J. De Langhuff & Co.*) 24s.

Mackenzie, Sir James. Principles of Diagnosis and Treatment in Heart Affections . . . . . (*Edward Arnold*) 7s. 6d.

McMechan, F. H. The American Year-Book of Anæsthesia and Analgesia . . . . . (*New York*) 40 c.s. 3

Maeder, A. E. The Dream Problem . . . . . (*Nervous and Mental Disease Monograph Series, No. 22*) cents.

Moore, C. C., and W. Partridge. Aids to Bacteriology. Third Edition . . . . . (*Baillière, Tindall & Cox*)

Morison, R. Surgical Contributions. Vol. I. General Surgery, 15s.; Vol. II. Abdominal Surgery, 25s. . . . . (*J. Wright & Sons*) 2 vols. 4 s.

Preston, E. A. The Healing Hand . . . . . (*Sunshine Mission to Sailors*)

Ross, G. W., and J. J. Loudon. A System for Case-Taking . . . . . (*Macmillan & Co., Ltd.*) 2s.

Short, A. Rendle. When to Advise Operation in General Practice . . . . . (*J. Wright & Sons, Ltd.*) 5s.

The Eighty-Ninth Annual Report of James Murray's Royal Asylum, Perth, 1915 . . . . .

The Third Report of the Indigenous Drugs Committee (Calcutta) . . . . . —

Transactions of the College of Physicians of Philadelphia. Vol. XXIII. 1915 . . . . —

University of Iowa Monographs. Vol. I., No. 1 . . . . (*Collected Studies and Reports*) —

Walker, Norman, and R. Cranston Low. An Introduction to Dermatology. Sixth Edition . . . . . (*W. Green & Sons, Ltd.*) 15s.

Weller, F. P. Tuberculous Pneumonia and Tuberculous Tuberculosis . . . . . (*Lea & Febiger*) net.

White, W. Hale. Materia Medica, Pharmacy, Pharmacology, and Therapeutics. Fifteenth Edition . . . . . (*J. & A. Churchill*) 7s. 6d.

EDINBURGH
MEDICAL JOURNAL.

THE UNIVERSITIES AND THE NEW ERA.*

By Sir ALFRED EWING, K.C.B., LL.D., D.Sc., F.R.S.,
Vice-Chancellor and Principal of the University of Edinburgh.

In responding to the invitation of the Faculty of Arts to deliver an address on this occasion, I am glad to know that I am following a precedent set by the late Principal. I hope it may often thus befall me to copy one whose memory is cherished with so affectionate a reverence.

It is with gratitude as well as pride that I find myself doing this first public act of service to our beloved Alma Mater. Like most of her children, I let go her skirts as soon as I could walk alone. I have wandered far and long, and now enjoy the traditional welcome of the returning prodigal. She has induced me to come—when, indeed, I was not thinking of it—by offering the fattest calf at her disposal. She has brought forth the best robe and put it on me—a robe in which Solomon might have challenged comparison with the lilies of the field.

This is no time for making merry, but one may be allowed to express a certain quiet gladness in renewing associations which have lost nothing of their power to stimulate and charm. It is forty-five years since I entered the University as an undergraduate. The memory is vivid of my first day of academic life. The scene was the address of a Lord Rector—Sir William Stirling Maxwell. Chosen of the students, and prepared to lay before them the ripe fruits of his scholarship, he found them a hostile mob who would not listen to a word he had to say. I asked why, and was told he had voted as they disapproved on what was the burning question of that day—the admission of women to study medicine. And now, when I take my place in the Senatus and the University Court, I find that the chief question for consideration is still the admission of women to study medicine. It would seem that the

* An address delivered at the Graduation Ceremonial of the University of Edinburgh, 11th July 1916.

mills of the University grind slowly. There is an absence of hustle which recalls the operations of geology or the evolution of species.

For nearly two generations the lady medicals, as they were called when I was a boy, have been wandering in the wilderness. Must the tale of weary years be extended, or is the land of promise now in sight? Let us hope that it is, and that they will enter the land and enjoy it without injury or loss to the existing occupants.

You of a later civilisation are no doubt less intolerant, or at least more courteous, than those who refused a hearing to the Lord Rector of 1871. But I trust and believe that the soul of undergraduate life remains as ardent as it was then, that to come up to the University still stirs every young spirit as it stirred mine, that the consciousness of intellectual expansion is still as glorious, that the springs of thought and feeling and action still respond as they did to the subtle influences of the place. Now, as then, Edinburgh makes its irresistible appeal to the æsthetic sense; now, as then, it fires the historical imagination. And if its students no longer have the good fortune to sit at the feet of a Lister or a Tait, teachers are never lacking who are worthy of that whole-hearted enthusiasm which gives the University its proper hold upon the minds and the affections of its sons.

The time has come for some of you to pass out into the bigger school of life. If you have studied to any purpose here, you will take with you the power to learn lessons which are awaiting you in that bigger school. You are to be congratulated on the successful finish of your academic career. The University has set her badge upon you; she owed you that for your sound work, and now you owe it to her not only that the badge be kept untarnished, but that you continue what she has begun. I mean something more than merely keeping up the habit of study. Remember that in her relations to the individual the University has three functions: first, to furnish the mind with knowledge; second, to quicken and widen the intelligence; and, third, to mould and set the character. The greatest of these is the last. It is by building well and truly on the foundations of character which the University has laid that you will best carry on her influence beyond these walls.

In the circumstances of the time you go out with a special inspiration. We live at present under the spell of a dominant motive beside which all the usual impulses of conduct are trivial.

You are about to play your several parts in the greatest struggle the world has ever known. There are some here who envy you the chance of such action as is the privilege of the young. But, young or old, the burden of the war presses on us all, and while we strive to carry it we are conscious of a new birth. The war has given life a seriousness of purpose which for most men was not there before. It has altered our standards of moral value. It has cut away rank growths which obscured the vision and encumbered the steps. It has given us a truer conception of social responsibility. It has swept aside much of what was mean, petty, sordid. It has convinced us of the primary duty of sacrificing self. It has made us face great issues with courage and sincerity: it has brought us back to the eternal difference of right and wrong. May I not say that in the personal experience of every man and woman here there is a sense of spiritual regeneration, that out of the misery and cruelty and madness of the war there comes a cleansing and uplifting of the heart?

> "There is some soul of goodness in things evil,
> Would men observingly distil it out."

Observingly or unconsciously we are all affected, and so profoundly as to give hope that the soul of goodness will prove more enduring than the evil from which it is distilled.

One's thoughts turn to those who in normal circumstances would have been with us to-day. The Universities have taken a big share in the war, but no more than a proper share, by sending of their best. Our reduced numbers are witness to this, and we are proud to think they are so much reduced. More than four thousand members of this University are serving with the forces: more than three thousand hold commissions as officers. There are many who can never come back. They have given themselves freely, even cheerfully. They have died — they are dying—so that what they and we hold most dear may live. They have found their kingdom, and who shall pity them? To those who do come back life can never be the same again. It will be coloured by the memory of adventure, it will be ennobled by the sense of accomplished renunciation. To them will apply the words the poet uses of the Happy Warrior:—

> "Whose high endeavours are an inward light
> That makes the path before him always bright."

May we not, then, turn hopefully and confidently to the future?

The influence of the war on our social fabric can be compared to an effect one sometimes sees in the study of metals. Let a piece of metal be exposed to certain conditions, and almost suddenly a great internal transformation occurs. Its external form shows no change. In the last analysis of the chemist it is still the same substance. The same molecules are there, but silently and invisibly they have marshalled themselves into wholly different patterns of structure. In a very real sense the material has become new. Its old attributes have disappeared. What was hard and brittle is now plastic: it may readily be shaped into other forms: it is capable of uses to which it could not have been put in its former state. The effect is a permanent one, persisting after the conditions which produced it have been removed.

Is there not some parallel to this in the new-made world that awaits us after the war, in the enormous changes—spiritual, social, political, economic, industrial, educational—that are even now taking place, and must increasingly take place in the early years of peace? When our civilisation, which is now straining to save itself, settles to the task of reconstruction, will it not find men's minds more plastic, traditions less controlling, prejudices less final? The community has adapted itself marvellously to war. It will be under an equally stringent obligation to adapt itself to peace, not in the old ruts, but conscious of new wants, new duties, new powers.

Amongst many other changes we may expect to see a far-reaching revision of methods and aims in education. If the war has demonstrated some of the good fruits of our schools and Universities, it has also laid bare defects which we must set ourselves strenuously to make good. It will lie with the Universities not only to act as guides of public opinion, not only to make their influence felt by the schools, but to see to it that their own house is in order. Is it too much to hope that they will show the plasticity which will be required of every element in the State during that anxious period of reconstruction, and that in the process they will not lose any of those essential qualities we venerate and love?

I am no prophet, believing with George Eliot that of all forms of human error the most gratuitous is prophecy. But it needs no prophet to affirm that the Universities will continue, as ever, to be sanctuaries of the spirit of man, where he may learn how to live not by bread alone. They will continue also to be temples

of research, prominences from which to gaze into the unknown, citadels from which to advance to the conquest of territory now unexplored.

But while these basic features remain, may we not look in the future work of the Universities for a more direct relation to industrial requirements and economic problems? The war has given a marvellous impulse to productive activity, but the current for the most part now floods a single channel, from which it must be diverted when the demand for munitions abates with the coming of peace. It should then be turned into other streams, fertilising and beneficent, in which it may swiftly re-create the wealth that has been destroyed, and also secure for the Empire sufficient independence in respect of all products that are vital to its strength. In this process the Universities have a great part to play. It will be for them to promote industrial efficiency, to revise the dogmas of political economy in the light of a broader ethic, and to give such teaching as may lead capital and labour alike to strive, in no spirit of antagonism, for an end which will need their sanest co-operation. Again, it will be for the Universities to bring science to bear on manufacture, to see that no barriers are allowed to come between the laboratory and the workshop, to prepare Theory for her honest nuptials with Practice. *Fas est et ab hoste doceri.* In these matters we have much to learn from the Germans. Students of the subject have known for years that we have been behind Germany in the technical application of science, and especially in realising the extent to which industry should be founded upon and guided by the results of research. The war has brought this lesson home to a wider public, and opinion is ripe for reform. It rests partly on the manufacturers and partly on the Universities to see that this handicap to national prosperity is removed.

No review, however brief, of the situation as affecting the Universities can omit to notice the place women are taking in public and industrial life. In their response to the nation's urgent call they have shown capabilities and adaptability beyond expectation. The range of their activities is no less remarkable than the devotion and courage which have levelled mountains of obstacle. Many of their new occupations must be transient, but it cannot be expected that all will come to an end. We may surely welcome the prospect of a permanent increase in the army of productive workers. If so, the responsibility of the University in providing higher education for women is enlarged, and it becomes more

than ever necessary to study how best to meet their special requirements.

In these and other ways the Universities of the future have tasks to perform which are worthy of their powers. It is probable that a larger proportion of the people than before will seek to obtain the higher education. It is certain that all will be less able to pay for it. This may throw on the Universities of Scotland a relatively large share of the common work, for their boast has always been that their doors are open to the poor. We cultivate the Muses on a little oatmeal. Our Scottish virtue of thrift, which provides that indispensable modicum for not a few deserving sons, has at last come to its own: its praise is on all the hoardings. It is an austere virtue, but an excellent one, and a powerful ally to education. More attractive, perhaps, and a still more powerful ally, is another national characteristic, the devotion to learning which for generations has permeated all ranks of our people, which made Scotland a land of universities when universities were rare. Long may that robust passion for knowledge bring to the University of Edinburgh, not from Scotland only, but from the Dominions beyond the Seas, a crowd of eager youths, destined in due course to pass out bearing torches kindled at our shrine. They will go their several ways: there is no corner of the Empire where they will not be found. The torch in each man's hand may in time serve to guide his fellows; it may spread illumination over some region, great or small, of the world into which it is borne. In any event, let him but keep it burning, and with its light about his own feet he will walk serene.

LECTURE ON ACETONURIA, ACETONÆMIA, AND ACID-INTOXICATION IN CHILDREN.*

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OFTEN when we are examining a sick child our attention is arrested by a sweet chloroform-like smell of acetone in the breath. This is sometimes very noticeable; but sometimes it may be overlooked, because it is partly obscured by an offensive odour as of decaying epithelium and secretions. In many of the cases a similar smell of acetone is noticed in the urine also.

Although these symptoms have long been observed, it is only

* Delivered at the Sick Children's Hospital on 22nd September 1916.

in recent years that light has begun to be thrown on certain aspects of their pathological chemistry and on their meaning from a clinical point of view. Our information on the subject is indeed, as yet, far from being complete or satisfactory, and on many points even the authorities are not fully agreed; so that it is not very easy for the physician to arrive at a confident and well-balanced view of the matter. Still, though much remains doubtful, a number of facts have been gathered which are not only theoretically interesting, but also of real value in practice.

In this lecture I shall try to lay before you what seem to me the most important of these facts.

When there is an abnormal amount of acetone in the blood we have what is called *acetonæmia*. This condition is worthy of attention, not because acetone is in itself harmful, but because its presence practically always implies that of the other two members of the acetone group—β-oxybutyric acid and diacetic (aceto-acetic) acid, and these are a real source of danger to health. The danger of their presence in the blood lies, we are told, not in any special properties they may possess, but merely in their acidity which changes the normal reaction of the blood. The word *acidosis* may be conveniently used to denote an acid state of the blood from whatever cause it may have arisen, and whether it is producing symptoms or not. When it has gone so far as to upset the patient's economy and produce morbid symptoms, we have the serious condition of *acid-intoxication*. The cases of acidosis with acid-intoxication which we meet with in clinical work are generally due to the acids of the acetone group; but similar symptoms have been produced experimentally in animals by various other acids, and there are also clinical types of acid-intoxication which have been found not to depend on the presence of acetone bodies but on that of other acid substances. For example, Howland and Marriott have recently [1] shown that the acid-intoxication which is not uncommon in some severe forms of diarrhœa in children is probably due to a deficient excretion of acid phosphates by the kidneys.

Causation of Acetonæmia.—The presence in the circulation of an abnormal amount of substances of the acetone group depends always on an increased fat-metabolism, along with an insufficiently rapid elimination of these products by the liver and kidneys. The increased fat-metabolism may, theoretically, arise from an excess of fatty matter in the food. It is, however, in most cases not due primarily to this but to an abnormal diminution of the

carbohydrates or to some interference with their digestion which renders them no longer available for utilisation by the tissues; for, while the usual carbohydrate metabolism lessens the splitting up of the fats, its diminution increases it. Constipation and starvation are found to increase fat-metabolism; and, clinically, it is found that they have the effect of increasing acetonæmia. The starvation alluded to may be brought about by lack of food, or it may arise from continued vomiting or diarrhœa, which have the same effect. The delayed excretion of the acetone bodies may depend to some extent on an idiosyncrasy, or it may be brought about by various passing conditions, such as constipation, sepsis, or the action of certain chemical poisons.

In addition to the diet-causes of acetonæmia there are certain forms of toxæmia which produce or aggravate it Examples of these are the poisoning caused by certain general anæsthetics, such as chloroform and ether, that due to salicylic compounds, and such septic absorption as takes place in appendicitis and from infected wounds.

Children are far more liable than adults to acidosis and acid-intoxication, and the younger the child the greater is this tendency. The presence of acidosis seems generally of no importance except in so far as it constitutes a threat of the graver condition. When acid-intoxication begins, however, it is always a serious matter, which calls at once for investigation and active treatment.

Symptoms of Acid-Intoxication.—When acid-intoxication sets in, the child becomes restless, irritable, and sleepless; he cries out and tosses about in bed, and is heedless of what is going on around him. This condition is apt to pass into one of definite drowsiness, with or without delirium; and in bad cases it may end in coma. Generally there is vomiting, which is sometimes urgent and uncontrollable; and when this is so, the vomited matter often contains a little blood. The bowels may be constipated, or there may be more or less diarrhœa with mucus, and perhaps blood in the motions. Some degree of jaundice occasionally occurs. The abdomen is flat, even retracted, and epigastric pain and some degree of abdominal tenderness are often complained of. In slight cases the tongue may be fairly clean, but it is often dry and coated. The pulse is rapid and soft. Toward the end in fatal cases, though acetone may still be noticeable in the breath, it may no longer be found in the urine, and there may be almost complete anuria.

The facies varies with the stage and with the severity of the illness. Sometimes it closely resembles that of grave abdominal disease, but in other cases, even when the symptoms are severe, one is struck by the entire absence of this appearance, at least in the early stages.

Much the most distinctive and interesting of the clinical phenomena in these cases is the peculiar exaggerated form of breathing, or hyperpnœa, which Kussmaul called "air-hunger." In this the respiration is not quickened to any great extent, and there is no cyanosis or other sign of respiratory obstruction; but the breathing is peculiarly deep and laboured, and it has more of a thoracic type than is usual in childhood. In most cases, both the breath and the urine smell strongly of acetone. The relative severity of the different symptoms varies greatly in different cases. When the acetonæmia occurs as a complication of severe organic disease in the abdomen, the symptoms are, of course, modified according to the condition present.

If the patient dies, the liver-cells are found to be either in a state of extreme fatty degeneration or otherwise seriously disorganised. The exact relation between this and the blood condition is not as yet fully determined. In one fatal case in which anuria had been present, extensive necrosis of the renal tubules was found with hæmorrhages into the glomeruli.

Test for Acetone and Diacetic Acid.—It is not now thought to be of any importance to test separately for acetone and diacetic acid. The best test for both is Rothera's. It must be applied when the urine is freshly passed, and is very delicate, as it detects diacetic acid in a concentration of 1 : 200,000, and 1 part of acetone in 20,000. It is as follows:—5 c.c. of the urine is mixed with an equal amount of a saturated solution of ammonium sulphate: 2 c.c. of strong ammonia is added, and afterwards a few drops of a 5 per cent. solution of sodium nitroprusside. If acetone or diacetic acid be present, the liquid acquires a rich purple colour and the length of the interval before the colour appears, and the intensity of the tint, afford some indication of the quantity of acetone and diacetic acid present.

There is, as yet, no clinical test available for the presence of β-oxybutyric acid.

Significance of Acetonuria.

It is not always easy to say at first what it means when a patient has signs of acidosis. Often it signifies very little indeed that

matters, but sometimes it is of serious importance. We may roughly divide the conditions under which this symptom occurs into three groups:—

1. *Physiological Acetonæmia.*—This may arise in some children merely from a temporary inadequacy of the metabolic processes. It may be set up by the child taking too little food, or by his food-stuffs being given in unusual forms or in unwise proportions —too much fat or too little carbohydrate—so that his chemistry has been deranged, although probably only in a passing and harmless way.

A curious variety of this sort of acetonæmia was investigated a few years ago by R. S. Frew.[2] He examined for acetone the urine of 662 children who were admitted to the wards in the Great Ormond Street Hospital, for all sorts of diseases and injuries, and found it present in no fewer than 408 of the cases (66·1 per cent.). The examinations were repeated daily for three days at least; and although acetone was almost invariably absent on admission, it appeared in the majority after the child had been 12 hours in hospital. It increased in amount for about 24 hours, and then diminished; and it ceased without treatment on the 4th day. Whether the child were ill or not, and if ill, what was the matter with him, made apparently no difference. The acetone was present more frequently and in larger amount the younger the child was. Whenever glucose was given, the acetone disappeared within 12 hours.

The explanation suggested to account for this temporary acidosis is that the metabolic processes are so unstable in childhood that they may be upset even by slight changes in the form of the carbohydrates taken. While the full significance of these interesting observations is by no means clear, there is no reason to think that this slight passing acetonæmia predisposes the child who has it to suffer from post-anæsthetic poisoning or any other dangerous form of acid-intoxication. In anxious cases, however, it is probably advisable, in case of mistakes, to delay operation if possible till the physiological acetonæmia has either passed off spontaneously or been stopped by the administration of glucose and sodium bicarbonate.

2. *Acetonæmia as a Complication.*—Acetonæmia often occurs as a secondary accompaniment and aggravation of some serious disease, owing perhaps to some toxæmia present or to the starvation which the disease or its treatment has occasioned. Thus it is met with in various sorts of septicæmia, in diarrhœa, peritonitis,

intestinal obstruction, severe vomiting from any cause, poisoning from phosphorus and other drugs, pneumonia, diphtheria, and many other fevers, tuberculous meningitis, spasmophilia, and so on. In these conditions the presence of acetonæmia often does not affect the diagnosis, nor generally, it seems, the prognosis, to any great extent; and the treatment is that of the original disease along with the administration of alkalies, laxatives, and easily assimilable carbohydrates.

3. *Acetonæmia as a Cardinal Symptom of Various Forms of Toxæmia.*—The detection of acetonæmia may be the first thing to reveal the presence of danger in one of the important forms of toxæmia of which acid-intoxication is a grave and cardinal symptom. In such cases the early recognition and prompt treatment of the acid-poisoning is all-important. The main clinical types of which this may be said are:—Diabetic acetonæmia, post-anæsthetic acetonæmia, salicylate poisoning, and recurrent or cyclic vomiting. There are probably various other types belonging to this group. For example, Abt has published[3] a series of cases of a very fatal type of acid-intoxication in young infants which differs in many respects from recurrent vomiting.

Diabetic Acetonæmia.

In diabetes the risk of acid-intoxication passing on to coma is particularly great in early life. When treating a diabetic child, therefore, we must constantly be on our guard and try to prevent its occurrence. For this purpose we have to take measures to ensure a regular action of the bowels, but at the same time check any tendency there may be to diarrhœa, and the urine should be kept mildly alkaline. We must also see that the carbohydrates in the diet are not too much cut down and that an excess of fat is not given (for example, in the form of cod-liver oil).

When signs of acid-intoxication appear, vigorous measures are called for. A purgative should be given at once, and also bicarbonate of soda by the mouth in doses of grs. xxx. every two hours in a tablespoonful of water, or, if the vomiting is severe, by rectum (℥ii. in 8 ounces of water every six hours). In extreme cases a few ounces of a 5 per cent. solution of sodium bicarbonate may be injected into a vein.* It is also advisable to increase the

* This solution must not be given subcutaneously, because, when it is sterilised by boiling, some of the bicarbonate is changed into carbonate, and this, though well borne by the veins, is very irritating to the subcutaneous tissues.

child's fluids by the subcutaneous injection of saline solution. An ounce or two of a 10 per cent. solution of glucose may be given by mouth or as an enema. The child must be carefully guarded from excitement and from all other causes of exhaustion.

Ordinary stomach sedatives are of little or no use in allaying the vomiting, but a small hypodermic of morphine is sometimes helpful. Of this gr. $\frac{1}{60}$ may be given to a child of 3 or 4, and gr. $\frac{1}{10}$ to one of 10 years.

Post-Anæsthetic Acetonæmia (*Delayed Chloroform Poisoning*).

In this country attention was first drawn to the danger of post-anæsthetic acetonæmia by Leonard Guthrie;[4] and further light has since been thrown on the subject by the investigations of Stiles and Stuart M'Donald,[5] Beesly,[6] Langmead,[7] and others.

The administration of chloroform, ether, or any other general anæsthetic always induces some degree of temporary acetonæmia. This occasionally leads to acid-intoxication, and is, therefore, not without danger even in apparently healthy children; and it may be fatal, unless the kidneys are able to respond to the increased formation of acetone bodies by a correspondingly increased excretion. Ether is much less harmful in this respect than chloroform, because, although it produces a somewhat greater degree of acetonæmia, it does less damage to the cells of the liver and kidneys, and therefore interferes to a smaller extent with their powers of rapid elimination.

The symptoms, which resemble those in other types of severe acute acid-intoxication, may come on within a few hours of the anæsthesia, or their appearance may be delayed for as much as three days. The amount of the drug used and the duration of its administration seem to have little or nothing to do with the occurrence of poisonous symptoms. If the urine contains acetone before an operation this is usually of no importance, provided the condition is not of the severe and acute type. Indeed, Beesly has made the interesting suggestion that the antecedent presence of chronic acetonæmia may even be of advantage, because it has accustomed the liver and kidneys to the elimination of acetone. If it is of the acute type, however, the danger is certainly great, especially if chloroform is the drug which has been used. When acetonuria and symptoms of acid-intoxication are found after an anæsthetic has been administered, the prognosis must always be guarded, for this form of acid-poisoning is a very

dangerous one. The risk is naturally much greater when septic conditions are present as in appendicitis, so that, in these, ether should always be preferred to chloroform. In all cases the idiosyncrasy of the patient has probably a large share in determining the degree of danger.

Treatment.—When such symptoms set in after the administration of an anæsthetic, the same measures are to be taken as in diabetic acetonæmia, and they must be begun energetically at once.

If there is any reason to anticipate danger from anæsthetic poisoning in a case, prophylactic treatment should be carried out. The following *precautionary measures* may be taken:—

1. For two or three days before the operation the patient should have bicarbonate of soda in sufficient doses to render his urine alkaline.

2. During the same time the fat in his food should be diminished as far as possible, and he should have plenty of carbohydrates.

3. A few ounces of a 10 per cent. solution of glucose may be given by mouth several times a day in place of less easily assimilable carbohydrates; and, two hours before the operation, an enema of the same solution.

4. The child must be carefully guarded from excitement, anxiety, and fright.

5. The urine should be tested for acetone; and if any is present it would probably be advisable to delay the operation until further examination has made it certain that the condition is not of an acute and dangerous type.

Salicylate Poisoning.

Generally salicylates, even in large doses, are well borne by children. The depressing effects that used to be feared from their use rarely occur, and, when they do, are not usually dangerous; and the headache and tinnitus aurium which adults often suffer from, are scarcely ever complained of. In some children, however, there is a real danger of poisoning from salicylates (or aspirin), and the symptoms produced are those of acid-intoxication—restlessness, vomiting, thirst, air-hunger, delirium, and drowsiness—and these may end in coma and death. In children who have this idiosyncrasy, quite a small dose of the drug is sufficient to produce the symptoms, unless proper precautions have been taken. The main precautions necessary are the careful

regulation of the bowels by aperients, and the administration of bicarbonate of soda along with the salicylate and in twice the quantity.

Treatment.—Whenever any signs of air-hunger and drowsiness set in in a child who is on salicylates the drug must be promptly stopped, a purgative given, and bicarbonate of soda continued in doses of grs. xxx. every hour or every two hours. In from 12 to 24 hours after the symptoms have subsided, the salicylate may be begun again in smaller doses, and gradually increased. When this is done the symptoms do not usually recur, even with fairly large doses. Evidently in this form of acetonæmia the liver and the kidneys are little if at all damaged and soon acquire the capacity to deal with an excess of acetone.

Recurrent Vomiting (Cyclic Vomiting).

This name is given to a fairly well-defined group of cases of obscure origin in which attacks of sudden severe vomiting recur at varying intervals, generally without any obvious cause, and are accompanied by acetonæmia, and occasionally by grave acid-intoxication.

Clinical Features.—In many cases the attacks, though distressing to the patient, give rise to no anxiety and have little effect on the child's health; and the mother, who has become accustomed to them, and probably calls them "bilious turns," may not even think it necessary to summon a doctor. In others the frequently returning sickness is so severe, and the patients are so seriously weakened by it, that for the time they become chronic invalids.

In a few instances the attacks, after beginning in the usual way, rapidly develop signs of acute acid-intoxication, and the child is soon in a state of great danger. These alarming symptoms may set in without any warning in children in whom former attacks have always been quite mild in character. This is at times so striking as to suggest the possibility that the entirely altered character of the illness must be accounted for by the presence of a complication which had not occurred on the other occasions. In one case, for example, a very severe attack of this kind seemed to be due to a coincident influenza which afterwards spread through the family.

The disease is mainly one of childhood, and in most cases the attacks are said to have begun in early infancy, or at least within the first three years. Sometimes, however, they occur for the

first time in the sixth or eighth year, or later. Although the children not uncommonly seem quite healthy in other respects, in many of the worst cases they are said to have always been delicate. They are not often obviously neurotic, and I have found in several a history of their having had asthma, urticaria, eczema, stammering, and other nervous complaints. In many of them uric acid crystals, or a copious deposit of urates, have been noticed in the child's urine. I have seen two instances in which patients who had suffered from pyloric stenosis in early infancy developed this disease in later childhood. This may have been merely a coincidence, but F. W. Shaw[8] and A. E. Russell[9] have published cases of recurrent vomiting in which pyloric hypertrophy was found post-mortem.

The disease is commoner in private than in hospital practice, and it occurs specially in the children of gouty and neurotic parents. Sometimes more than one child in a family is affected. The patients are rather more frequently girls than boys.

The intervals between the vomiting attacks vary greatly in duration in different cases, and sometimes also in the same case at different times. They may for a time return as often as every fortnight, more often every one, two, or three months; sometimes they occur only once or twice a year. The attacks may continue for many years, but generally they tend to cease as the child grows older and stronger.

Symptoms of the Attacks.—In many instances the attacks set in without any warning. In others the mother is forewarned by the child becoming pale, tired-looking, and irritable, yawning a good deal and refusing food. Sometimes there is flushing, but more often a feeling of chilliness is complained of. Occasionally there are local symptoms, such as headache, or pain in the neck or limbs, or in the pit of the stomach. In one or two of my cases there was antecedent diarrhœa; much more frequently, however, the patient had been troubled with constipation; flatulence is a common symptom. When such indications of a threatened attack occur, they may last for from 12 to 24 hours before the actual vomiting begins.

The vomiting, when it comes, is often very severe and distressing; and everything, even small quantities of water, may be rejected. The ordinary treatment of dyspeptic vomiting has no effect. At first the vomited matters consist of food, mucus, and bile; and, later, there is often some blood in it. The child is drowsy, restless, and thirsty, and may complain of severe headache.

The temperature sometimes remains normal, but generally it rises to 101 or 102 at least, and it often reaches 104 or even 105. There may be vague abdominal pains, but no definite localised tenderness is to be made out. The abdomen, in uncomplicated cases, is flat or retracted. Occasionally there is diarrhœa, but generally obstinate constipation. The tongue is furred, and the pulse weak and rapid; the patient usually looks very ill. Acetone is generally, if not always, to be detected in the breath and in the urine at some stage of the attack. The urine may also contain a small quantity of albumin or blood and a few hyaline casts. Rarely there is a degree of jaundice.

The vomiting and other symptoms generally subside, often suddenly, about the third day, but they may continue for six or seven days, or longer.

The serious cases, in which acid-intoxication appears, closely resemble those of diabetic acetonæmia, and they generally respond to the same treatment, but a large proportion of them end fatally. I have had the misfortune to have had four cases of this kind, which died in spite of energetic treatment. One of the patients was 18 months old, one 2 years, and the others both about 5 years.

Occasionally cases are met with in which there is recurrent pyrexia with or without headache, and with little or no vomiting. These cases are probably of the same nature as those of recurrent vomiting, and they should be treated in the same way.

Causation.—The pathology of recurrent vomiting is still obscure. The view at present held is that it depends on a "poisoning arising from the intestinal tract with resulting imperfect oxidation of the fats and their accumulation in the liver."[10] It is important, however, to emphasise the fact that it is in children of specially neurotic constitution that this is apt to occur; and we shall also probably not be far wrong if, with Holt and Rachford,[11] we regard lithæmia as having a good deal to do with the causation of the disease. This view is often supported by the result of treatment. There seem to be no good reasons for suspecting morbid conditions of the appendix as a frequent cause of the attacks, for I have known several cases in which, after its removal, the symptoms continued recurring just in the same way as before the operation. The cases in which serious symptoms set in, seem always to be due to acid-intoxication, and this usually, if not always, depends on acetonæmia.

Definite exciting causes of the attacks are generally absent so

far as can be observed clinically; but, now and then, we find that their recurrence has been precipitated by a chill, an indiscretion in diet, some form of exhaustion or over-exertion, or a feverish illness such as influenza.

The disease seems, in most ways, to be closely allied to migraine. A history of sick headaches, asthma, and eczema, or of vomiting attacks, in the parents or near relatives, is not uncommonly obtained, and I have known a number of cases in which the children themselves developed migraine in later life. In this disease also, as in migraine, there is often a family history of gout, as well as of various other manifestations of an unstable nervous system.

Prognosis.—As has already been indicated, this is generally good, but in view of the possibility of acid-intoxication occurring, it must always be guarded. There is a considerable likelihood that the attacks may continue to occur for some time; but, generally, under treatment they diminish in frequency and severity, and they often cease altogether. As has been previously mentioned, there is a tendency to spontaneous recovery about the time of puberty, if not before, though the symptoms may then pass into those of ordinary migraine.

Diagnosis.—A first attack is apt to be mistaken for one of meningitis, of acute gastric catarrh, or of intussusception. Attention to the history of the case and to the symptoms should generally suffice to exclude these conditions. Other diseases which sometimes closely resemble recurrent vomiting are chronic or relapsing appendicitis and some forms of abdominal tuberculosis. In these also the history of the case is helpful, and the presence of tenderness and other local signs is usually sufficient to enable a correct diagnosis to be made. The mere presence of acetone in the breath and urine is not in itself of much assistance, as it is so often found in other morbid conditions with vomiting.

Treatment.—When an attack begins, the patient must be kept in bed, and the bowel should be emptied by a purgative, and, if that is vomited, by an enema. In most cases decided improvement follows the administration of bicarbonate of soda in large quantities (120-180 grs. or more in the day), as was first pointed out by Edsall.[12]

So long as the violent vomiting continues, all food by the mouth, including liquids, should be withheld. Sometimes, however, after the attack has lasted for a while, a soothing and settling effect may be obtained from washing out the stomach or from the administration of a large drink of warm water and soda, which is generally vomited immediately. Occasionally, if there

is no suppression of urine, an injection of morphine may be of service. Saline enemata, if retained, have the great advantage that they relieve the distressing thirst from which the patient suffers, and encourage the action of the kidneys.

During the intervals between the attacks a great deal can often be done to lessen their frequency and severity. The diet should be carefully regulated and all over-eating prevented. Excess of fat in the food must also be avoided; little butter is to be allowed, and no cream; in bad cases the patient's milk had better be skimmed. Green vegetables and fruit in moderation are generally beneficial. A cupful of hot water, on waking in the morning, with or without the addition of Vichy water, is often useful.

Great improvement sometimes follows the use, for long periods, of salol (grs. iii.) or salicylate of soda (grs. iii.-vi.) with bicarbonate of soda (grs. v.-x. according to age). These should be given with plenty of water thrice daily, about an hour before meals, and should be continued for weeks. The regular action of the bowels must, of course, be most carefully attended to. In some cases this treatment has no good effect, but I think that it is always worthy of a careful trial. It may very naturally be asked whether salol and sodium salicylate, even in such small doses, might not aggravate the tendency to acid-intoxication. I have, however, given these drugs so often in cases of recurrent vomiting with apparent benefit, and never with any bad result that I know of, that I feel sure that there is no risk in their use in this disease. The acidosis of salicylate poisoning is less dangerous than most other severe types of the condition; and it seems quite possible that the administration of small doses of such substances may not only do no harm, but may even be helpful by training the liver and kidneys in the elimination of acetone bodies.

In addition to dieting and the use of medicines, it is always important, if possible, to avoid all causes of exhaustion, such as prolonged excitement or a long railway journey.

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THE HISTOLOGY OF DISSEMINATED SCLEROSIS.

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Part II.

Histological Study.

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(1) Introduction.

It has already been mentioned that this study is based on an extended pathological observation of nine cases of disseminated sclerosis. The brain and spinal cord from most of these cases were equally thoroughly examined, but in only one instance was there the possibility of following the case clinically and anatomically. That case, therefore, has been taken, more or less, as a

standard of comparison, and it is proposed to begin this part of the paper by giving a short account of the clinical history of this patient. In the Appendix to the complete paper will be found the available clinical notes and post-mortem reports in the other cases, as well as an account of the general histological features and topographical distribution of the areas of sclerosis in Weigert-Pal sections.

Clinical History.

L. W., aged 28, a kitchenmaid by occupation, was admitted to the late Dr. Alexander Bruce's wards on 4th April 1910, complaining of weakness in both legs, inability to walk, and tremors in both arms and legs, the duration of these symptoms being about two years.

History of Present Illness.—At the end of March 1908 the patient, who was then employed at one of the baths in Edinburgh, was reaching up to clean the wall with a long brush, when she stepped too far back and fell into the deep end of the bath. She was pulled out immediately, but was, of course, wet through to the skin. This gave her a great fright, and she screamed for about ten minutes. When she had quietened down, she had a hot bath, put on dry clothes, and went home. She said also that a menstrual period came on about two hours before she fell into the water; after this it completely stopped and did not return until a fortnight later. The above accident took place in the forenoon, and by the afternoon she felt so much better that she returned to her work.

She continued at her work until the end of June, when one morning she found that both her legs had become swollen and tender, that she had shooting pains down her right leg, and that she had great trouble in rising out of bed. She sent for her doctor, who thought the symptoms were due to muscular rheumatism, and treated her accordingly. She was slightly feverish at this time and remained in bed for a week, and at the end of a fortnight returned to work. She noticed, however, that she did not seem to be able to walk so quickly as previously, that she was slightly lame, and that her right knee was stiff.

She worked steadily at the baths until March 1909, when she began to find her work too heavy, and she accordingly took an easier situation as kitchenmaid at a golf club house. She succeeded so well at this that after a month she became waitress. After four months this post became too heavy for her, and she returned to her old post of kitchenmaid, but two months later had to give this up also, and has done no work since 12th November. Two months before admission her symptoms had become more marked. Both legs became very stiff, and she could only walk with the help of a stick. Her arms

became shaky, and one of the reasons why she left the club-house was that if anyone gave her a sudden start her arms became very shaky, and once or twice she dropped a tray full of dishes. Although usually constipated, she was subject to sudden precipitate action of the bowels as well as to incontinence. Her friends had noticed that she had been getting very slow in her speech, and that sometimes she had difficulty in pronouncing words. The menstrual periods had been somewhat irregular, usually occurring too frequently, but lasting only a very short time. As these symptoms became progressively worse she was sent to the Royal Infirmary, Edinburgh, to be examined by the late Dr. Alexander Bruce, who at once admitted her to his ward.

Previous Illnesses.—Patient had always been remarkably healthy as a girl, and, with the exception of occasional "bilious headaches," can remember of no other illnesses.

Condition on Admission.—Her appearance was that of a strong, healthy girl with a good fresh colour. Smell and taste were normal. The pupils were circular and equal, and reacted both to light and to accommodation. The eyes were freely movable in all directions. There was slight lateral nystagmus on looking to the left and slight rotatory nystagmus on looking upwards. Both discs were normal.

Sensation to touch, heat and cold, and pain was intact; and there was some tenderness on deep pressure of both calves.

The knee-jerks were both exaggerated, the left rather more than the right. Both Achilles jerks were also exaggerated, and Babinski's sign was positive on both sides. The abdominal and upper limb reflexes were all present and apparently normal. There was no wasting of any muscles. There was slight inco-ordination in the finger-nose test with the right hand, but no intention tremor and no inco-ordination of the lower limbs.

The heart was not enlarged, the apex-beat not displaced, and the pulse was 70. There were no murmurs present, and the arteries were not thickened nor the blood-pressure raised.

The chest was well formed and the expansion good. Neither percussion nor auscultation revealed anything abnormal.

There was no enlargement of the liver or spleen. The stomach was not dilated, the digestion was good, but there was considerable constipation. The urine contained neither albumen, sugar, bile, nor pus, the specific gravity being 1017. She has occasional attacks of precipitate micturition.

Progress.—She was put to bed and at first made good progress, but on 25th May numbness in the left arm developed suddenly. On the 29th she felt deaf in the right ear, with dull buzzing sounds, and noticed that her right eye watered exceedingly. She complained of feeling her face squint on speaking. On examination a right facial paralysis was found. The right eyelid scarcely moved on attempting

to close the eye. The electrical reactions on the affected side of the face were normal both to Faradism and to galvanic stimulation. On the 3rd of June she developed diplopia on looking to the right, and was found to have a paralysis of her right external rectus. The other movements of the right eye were normal, and the left eye was unaffected. The pupils were equal, rounded, and dilated, and reacted to light and to accommodation sharply. Five days later the tongue was seen to protrude to the left side, and slight difficulty in speech was observed, which was followed two days later by difficulty in swallowing. Dr. J. S. Fraser, who at this time examined the ears, reported that both vestibular apparatuses were functional, although possibly the activity of the right was slightly diminished. By the 8th of August the weakness in the legs had become very marked and they were painful on attempting any movement. Spasticity in the legs soon developed. On 14th August severe vomiting set in, which was scarcely affected by any treatment. On the evening of 15th August she complained of dimness in seeing, and next morning was found to be blind in both eyes. On examination the discs showed some pallor but no neuritis. Muscular wasting now developed and proceeded rapidly, especially in the legs. The face became more hollow and sunken, diarrhœa and vomiting set in, and a catheter had to be passed every eight hours. From now onwards she went rapidly downhill, sometimes slightly better and sometimes slightly worse. She had an extremely septicæmic look, and died suddenly and unexpectedly on the morning of the 5th of September, after having passed a better night and had a little breakfast.

Post-mortem Report.

Brain.—No clots in superior sinus. Convolutions atrophied. Some general opacity of pia arachnoid. Fluid in subarachnoid space.

Cord.—Shows irregularly scattered areas of bluish-grey colour varying in size and shape. Cord as a whole is small. Similar areas in pons and medulla.

Macroscopically the cerebral and spinal meninges were in the main normal, but in places where the grey and greyish-blue areas in the cord reached the surface the pia over these areas appeared slightly opaque. On section of the cord at various levels, numerous gelatinous grey areas were found, and also parts of a softer consistence with a whiter colour than the normal tissue of the cord. Sections of the medulla oblongata and pons indicated the same two types of areas, parts of the pons appearing so affected that there were only islands of normal tissue, and the floor and immediate neighbourhood of the 4th ventricle were also markedly involved. Numerous areas were found in the horizontal sections of the cerebral hemispheres at various

levels, not only in the basal ganglia and white matter, but also in the cortex and subcortical white matter. There was a well marked periventricular sclerosis, especially of both the posterior horns of the lateral ventricle extending down into the descending horn on both sides. The ventricles were not dilated, and their surface was smooth but cloudy, and raised in slight ridges corresponding to the venous branches which are present underneath the ependyma, especially at the posterior and anterior horns of the lateral ventricle. These veins were distinctly outlined and surrounded by a zone of gelatinous tissue.

The size of the areas varied very considerably: in the cord it was impossible to define macroscopically, either on the surface or on section, any of the areas, as they seemed to run into one another. In the brain, however, isolated areas were the rule, though here also irregular areas of different size and form coalesced. Those isolated in the white matter varied in diameter from 1 to 10 or 12 millimetres, and were mostly oval or circular. The largest were found in the immediate neighbourhood of the roof of the lateral ventricle, and gave the impression of being upward extensions of the periventricular sclerosis of the roof.

In examining the microscopic findings it is important to recognise that different stages of the disease come under observation, and it would appear natural to differentiate acute, subacute, and chronic stages, for they are often enough found close together in one and the same case. The finding at the autopsy of patches at different stages in their evolution accords with the clinical history of the affection, which has usually progressed with remissions and relapses. It is, however, by no means universally admitted that the recent areas are transformed into those which would seem to represent a chronic process, in which all traces of inflammation have disappeared. If such chronic areas do not develop on the basis of the former, it must be acknowledged that there is a strong justification for looking upon them as a multiple gliosis, related to an anomaly of development, and upon the recent areas as an acquired form of sclerosis, whose origin lies probably in toxi-infective conditions.

This study, therefore, begins with a short description of these two types of areas; the one an old area, typical of the "sclérose en plaques" of the earlier writers; the other, a recent area, corresponding to those found in the so-called acute multiple sclerosis of recent writers.

(2) Structure of Different Types of Areas.

1. *An Actual Sclerotic Area.*—(a) *In the Spinal Cord.*—The histological picture of such an area, *e.g.* in the posterior columns, cut in the longitudinal direction of the nerve fibres, is a very simple one (Figs. 50 to 52). It consists almost entirely of newly-formed fibrils which are arranged parallel to the original course of the nerve fibres. In Weigert sections, counter-stained with picro-fuchsin, we recognise at once, under a low magnification, three of the outstanding characteristics of an old sclerotic area: the disappearance of the myelin sheath of the nerve fibre, the proliferation of the glia, and the alterations of the blood-vessels. By means of a glia stain and a diffuse stain these changes are confirmed and their details revealed; by an axis cylinder stain the fourth histological characteristic is represented — the persistence of numerous axis cylinders: and by the Marchi method it is seen that this old sclerotic area contains no degenerating nerve fibres nor granular cells containing the products of the degenerated myelin.

The shape of this area is brought out clearly by Weigert's stain as an elongated oval with its long diameter in the long axis of the cord. The continuity, therefore, of the nerve fibres is broken by this oval area, and the absence of the myelin at the sides is more or less sharply defined from the surrounding tissue, the margins forming more or less sinuous lines, but the limits of the area, on longitudinal section, are never straight or even curved outlines, for individual nerve fibres or bundles of such pass into the borders. The myelin sheath of these fibres stains irregularly and weakly, and the termination is usually broadly broken off, or it may be swollen or narrowed.

Numerous fine glia fibrils occur in regular, undulating lines parallel to and close to one another (Fig. 51). The neuroglia nuclei lie often in short rows or in groups, but more often, as in this area, isolated with the long diameter in the long axis of the fibrils, and, as a rule, quite independent of them. The number of the nuclei is smaller than in the normal tissue, but in structure and form they are slightly larger, clearer, and more oval. Between the glia fibrils and surrounded by them are found numerous axis cylinders, which are mostly thin and fine but thicker and more homogeneous in structure than the glia fibrils (Fig. 51). They do not run in such regular lines, and frequently show as faint diffusely-stained bands. The impression is often given that in place of the myelin sheath there has been a substitution of glia fibrils, forming a glious sheath to the axis cylinder. Lapinsky has wrongly interpreted this process of substitution as a metamorphosis of the medullated sheath—a conception which accentuates the enveloping character of the glia fibril proliferation.

The blood vessels stand out clearly in this sclerotic tissue. Those

cut transversely show thickened, homogeneous, pink-stained walls, and on longitudinal section the numerous longitudinally-running small vessels (Fig. 52) have a similar structure, with few cell elements even in their adventitia. The lumen of the smallest capillaries is often obliterated and that of the larger vessels narrowed. Weigert picro-fuchsin sections show beautifully the arrangement and distribution of these smaller vessels and bring out the abundant vascular supply of the various tissues.

At the periphery of the area, especially at its upper and lower limits, diffuse stains bring out a zone where intensely stained nuclei are in great abundance: these nuclei, in iron-hæmatoxylin stain, are seen to occupy the spaces between the nerve fibres projecting into the sclerotic tissue, and their increase can be traced for a small distance among the fibres of the normal tissue on all sides. The glia fibril proliferation and thickening of the vessels are here also evident, and this zone can be looked upon as a zone of transition between normal and sclerotic tissue—a zone where the pathological changes gradually cease till normal tissue is reached.

When the glia proliferation has taken the simple course represented in the former paragraph, a transverse section of such an area shows the glia fibrils as minute fine points which replace the normal glia meshes and the space originally occupied by the myelin sheath, and which more or less surround, as a ring, larger, more diffusely stained, and more homogeneous points—the axis cylinders.

The impression is received that here again the normal architecture of the tissue is preserved; that the myelin sheaths have disappeared, and have been replaced by a fine close fibre formation; that the walls of the blood-vessels, even the small capillaries, have thickened walls with few cell elements; that the axis cylinders have, at least to a considerable extent, been retained; that there are present in the area or its periphery no indications of the degeneration of myelin (Marchi method), thus allowing it to be supposed that the process which caused the myelin destruction has come to a standstill; and, finally, that there are evidences at the periphery of the area, in a nuclear proliferation and diminution in myelin fibres, of a process which has left these traces of a reaction to its further progress.

These areas, just described, in longitudinal and transverse section, are typical of the sclerosis which is the essential substratum of the disease known as disseminated sclerosis. They show a compact glia fibril proliferation, apparently without spaces, for only here and there are there traces of the original glia meshes. The area of dense sclerosis is regularly surrounded by a peripheral transition zone, which interposes between it and the healthy tissue. This transition zone shows very numerous nuclei of small and large glia cells, with more or less developed glia trabeculæ.

(b) *In the Cerebral White Matter.*—In comparing such an area with a similar one in the spinal cord, the first points to be noted are (1) that in the brain it is usual to get a more defined outline of the patch; (2) that a central vessel cut transversely or longitudinally is more frequently present; and (3) that the persisting axis cylinders are fewer and show more evident indications of a previous involvement. The affected area, in Weigert and Marchi sections, stands out much lighter in colour than the surrounding tissue, giving a negative picture, in marked contrast to the positive picture of the condensed and darker staining tissue in glia sections (Figs. 65, 66).

If the sclerosis affects a portion of the brain substance in which the nerve fibres run normally in very varied directions, *e.g.* in a small defined area in the central white matter above the roof of the lateral ventricles, the resulting tissue is a very dense network in which the original glia spaces are replaced by fine fibrils. These fibrils are very unequal in size; the larger form larger meshes into which the finer fibrils cut and make finer meshes, and the tissue becomes denser and denser till the meshes are inconceivably fine (Fig. 56). The size of the glia cells varies very much, but the body of the cell is in general round or slightly elongated, and may contain one or more nuclei but little protoplasm. Around the blood-vessels, even the capillaries, is a zone in which the fibril formation is even more dense; the fibrils rarely form a "corona ciliaris," but, rather, concentric layers of fibrils closely pressed together, with few nuclei, form an outer dense glia sheath to the vessel (Fig. 69).

Such cerebral areas, therefore, also show that the change consists in demyelination; a complete substitution of the spaces by fibrillar tissue, which in its arrangement forms simply a condensation of the original glia network; a thickening of the walls of the vessels originally present in the tissue; and, finally, the formation of a narrow but dense nucleated peripheral zone, which forms the transition to normal tissue.

2. *An "Early" Area.*—(a) *In the Spinal Cord* (*cf.* Figs. 41, 53).—The histological picture of such an area, *e.g.* in the posterior columns, with the nerve fibres cut transversely, is a very complicated one. Weigert sections show an irregular non-medullated area which has formed with the posterior median septum and its vessels as a centre. At the periphery, on all sides, isolated nerve fibres pass for a short distance into the area, and, under high power, it is seen that their myelin ring is very thin, or stains diffusely and shows evident signs of degeneration. The tissue of this area, even in Weigert sections, is seen, notwithstanding the absence of myelin and the presence of changed vessels, not to correspond to the dense sclerotic fibrillar tissue seen in the former area described. The blood-vessels—arteries, veins, and capillaries—are all dilated and engorged with blood, and their walls,

instead of the homogeneous, thickened, structureless tissue, show in the intima and media little recognisable change from normal: but the adventitia has its lymphatic spaces dilated and filled with nucleated elements, and these same nucleated elements are scattered irregularly through the non-myelinated tissue, and in the blood-vessel walls of the transition zone.

The most characteristic features of this area are seen best with diffuse stains, which show the presence of a very large number of nucleated cell elements with a considerable amount of protoplasm. Most of these large cell elements correspond roughly to pathological examples of the proliferated spider cells of the normal tissue (Fig. 53*a*). From the protoplasm radiate in all directions very fine branching, protoplasmic processes, which break up into a fine network. Many of these cells are multi-nucleated, and vary greatly in shape and size from star-shaped forms to those with crescentic outline and bi-polar forms (Fig. 57). Their processes bear very frequently a very definite relationship to the vessel walls—a relationship which will be more fully emphasised when describing recent areas in the cerebral white matter (Figs. 58 to 60). Smaller and darker-stained nuclei, with little protoplasm and no processes, can also be found in considerable numbers. In the spaces formed by the large branching processes of these cells and in the adventitial spaces of the blood-vessels lie the second tissue elements characteristic of such an area. These are larger, rounded, nucleated cells, with a large amount of vacuolated protoplasm: they correspond to the phagocytic cell of the central nervous system, and their vacuolated appearance is derived from the solution of the fine degenerated myelin granules in the process of hardening. Nissl has given to these cells the name "Gitterzellen" from their morphological appearance, but they are more generally known as compound granular or fat-granular cells ("Fettkornchenzellen") from their function of absorbing the products of the disintegration of the myelin. These cells lie not only in all the tissue interstices, surrounded by the branching processes of the glia cells, but in the lymphatic spaces of the adventitia of the blood vessels—whither they have in all probability been drawn in from the tissue spaces by the suction influence of the lymph flow in the adventitial lymphatics. Round even the smallest capillaries these cells form a complete ring, and the area under low power assumes a very characteristic appearance. The presence of such large numbers of fat granular cells gives to the blood vessel walls an appearance of a cell-infiltration, for in the vessels larger than the capillaries they are present in several rows, often closely compressed. In addition to these cell elements in the adventitia other nucleated elements add to the nuclear abundance of the vessel wall. The endothelium, especially of the capillaries, frequently shows evidence of a distinct proliferation, and in the adventitia are found dark-stained

nuclei together with nuclei of a vesicular clearer character, both of which have probably arisen from the proliferation of the cells of the adventitia and the endothelial cells of its lymphatic spaces. Diffuse stains bring out, although feebly, one further tissue component—the axis cylinders. These are no longer the sharply defined, homogeneous points of the dense sclerotic tissue, but a faintly-staining, almost unrecognisable, swollen structure, which is a lesser degree of the intense swelling of the axis cylinders seen in marked œdema of the cord. These swollen axis cylinders are usually found lying in the tissue meshes, with no trace, or only slight traces, of myelin around them, and often closely compressed between the fat-granular cells and the protoplasmic processes of the large glia cells. There is as yet almost no attempt at fibril formation on the part of these glia cells, although the processes at their lateral margin and terminations show a definitely darker staining.

It has been seen that Weigert sections show the almost complete absence of myelin in this area; that diffuse stains give (1) the characteristic appearance of the two cell elements—proliferated glia cells with numerous processes and the fat-granular cells; (2) the numerous dilated blood-vessels: and (3) the persistence of numerous swollen axis cylinders; and that glia stains give as yet no definite fibril formation. There remains now to be mentioned the appearance with Marchi-stained sections (Figs. 41, 42). This is the most characteristic of all, and has given to the areas the name of "fat-granule cell myelitis." Each of the large vacuolated cells in the tissue spaces and walls of the blood-vessels is found to be composed of a very large number of minute granules of a substance staining black with osmic acid. Most of these granules are quite round, but a few show irregular contours from compression. Granules are found also in the spaces between the cells. Under low power the area is thickly studded with these black granular cells, which also form concentric rings around the blood-vessels. No trace of normal myelinated fibres can be found within the area, but in the transition zone are recognised numerous fibres in all stages of degeneration. In this transition zone, in addition to numerous degenerating myelin fibres and vessels with rows of fat-granular cells in their walls, we find also a marked proliferation of the glia cells and protoplasmic processes, and a widening of the normal glia meshes, but the degeneration of the myelin has not advanced to the stage of complete disintegration and its absorption by cells. The glia meshes of the adjoining normally myelinated tissue are distinctly widened, the cells also enlarged, and the blood vessels engorged and dilated

Longitudinal sections (Figs. 47, 48) of such an area are very valuable in showing the changes in the nerve fibre as it passes into the area: the characteristic appearance of long rows of fat-granule cells, which seem to occupy the spaces left by the removal of the degenerat-

ing myelin of the nerve fibre, and also the longitudinal running vessels surrounded by elongated layers of similar cells (Fig. 43). Diffuse stains show the large glia elements already referred to, lying often in rows with the nuclei of two adjoining cells lying close to one another, as if they had arisen from the division of one cell (Fig. 57). The long, branching, protoplasmic processes extend around, and almost envelop, the rows of fat-granule cells, and in some parts the direction of these processes is already becoming longitudinal (Fig. 48). Almost pushed aside between the rows of fat-granule cells and the large glia cells with their protoplasmic processes can be found faintly stained, homogeneous, swollen bands, which represent axis cylinders (Fig. 48), and here and there are large numbers of rows of finer and larger granules staining with the same tone as the bands. These are the remains of the disintegrated axis cylinders, which seem, after the stage of severe swelling, to become broken up into large granule formation and gradually into finer granules before they ultimately disappear.

"Early" areas in the cerebral white matter show the changes in the cell elements at their most distinctive stage of development.

"Early" areas, therefore, consist largely of closely arranged fat-granule cells between which lie the large protoplasmic, proliferated glia elements; of dilated vessels, with fat-granule cells and other nucleated elements in their adventitial spaces; of markedly altered persisting axis cylinders; and of a gradual transition zone in which these changes are less marked and in which degenerating myelin fibres may be found.

If now the old sclerotic areas are briefly contrasted with those which have been termed recent areas, it is found that the areas typical of the "sclérose en plaques" of Charcot are marked by the following histological characteristics: the complete absence of myelin (Weigert stain); the presence of a dense fibrillar tissue (glia stain); the persistence of numerous axis cylinders (silver impregnation method); the presence of numerous blood-vessels with condensed, sclerosed walls (diffuse stains); and the complete absence of any evidence of myelin degeneration (Marchi method); and, finally, a nucleated transition zone, which gives frequently an abrupt passage to normal tissue. On the other hand, the recent areas, while presenting a striking contrast to the former, have also some of these characteristics, but in a lesser or different degree: the absence of myelin within the area; a very slight commencing glia fibril formation, but a very intensive glia cell proliferation; the persistence, to a much less extent, of axis cylinders and those persisting being markedly altered; the

presence of numerous dilated blood-vessels with nucleated elements in their adventitial spaces; the marked indications of a previous myelin degeneration in the presence of fat-granule cells which fill up all the interstices of the tissue and the adventitial lymph spaces of the blood-vessels; and, finally, a very gradual nucleated transition zone in which all these changes are less marked, but are combined with an evident degeneration of the myelin of the nerve fibres in this zone.

The question naturally arises: What relation do these two types of areas bear to one another? The finding of both types side by side in the same case, and the clinical history—with its remissions and relapses—argue for a close connection between them. Yet numerous writers, while not denying that the end result of the recent areas is a sclerotic tissue which bears a close agreement to the old sclerotic areas typical of the disease first designated by Charcot disseminated sclerosis, claim that true or primary disseminated sclerosis arises solely on the basis of areas of the first type, in whose evolution there is no stage corresponding to that of "fat-granular cell myelitis." Areas of sclerosis which pass through the stage of the second type must be designated as secondary sclerosis, and those areas arise on the basis of a disseminated myelitis.

The questions must then arise: What is the evolution of the first type? and what are the stages in the evolution of the so-called secondary sclerosis? In the next section an endeavour will be made to trace their respective evolution.

3. *Evolution of an Actual Sclerotic Area.*—A. *Through Stages of Increasing Glia Hyperplasia.*—The supporters of the view of a primary form of disseminated sclerosis claim that the essential lesion lies in the neuroglia tissue. By some anomaly of development this tissue, in certain areas, undergoes an intensive proliferation, which, by a gradual constriction of the glia meshes, produces a slow atrophy of the myelin sheath of the nerve fibre. The myelinated nerve fibre, therefore, in certain areas, undergoes a progressive reduction in its volume till it is completely replaced by the glia fibril proliferation, leaving the axis cylinder preserved. This proliferation goes a stage further than a mere substitution of glia fibrils for myelin sheath, for it is of such intensity that in no other condition is the glia proliferation so marked as in disseminated sclerosis (Weigert). It is claimed that not only is there a direct action on the nerve fibres by direct compression of the proliferating glia, but that there is an indirect action upon their nutrition. By accumulating around the vessels the glia, if it does

not close their lumen, is said at least to limit their expansion and diminish the blood flow, and interfere with the circulation in the perivascular lymphatics. In this simple atrophy of the myelin sheath the degenerated products, owing to the slowness of the process, are removed just as formed, in the form of very fine granules or in solution, without requiring the presence of true granular cells. These cells are said to appear only when the process is more rapid: they may occur at the periphery of an area of true primary sclerosis, where the affected tissue causes a reaction in the normal tissue and an excentric spread of the process occurs.

Weigert sections of such an area show a complete absence of myelin in the fully sclerotic tissue, but, in the earlier stage, the myelin sheath appears thin and atrophic and is gradually lost. The axis cylinders may be recognised for a long time apparently little altered in quantity or quality, but, later, they atrophy and finally disappear or fuse with the glia tissue. The blood-vessels present condensed walls with few cell elements. With glia stains the gradual thickening of the glia trabeculæ, and of the finest fibres forming the reticulum, can be followed up till the glia meshes are almost obliterated and the dense fibrillar felt-work takes their place. Simultaneous with the thickening of the glia trabeculæ and reticulum the glia nuclei in the nodes of the reticulum and within the trabeculæ have enlarged and proliferated, becoming large protoplasmic cells with long ramifying processes which join the general glia reticulum.

Such areas, from the slowness of the demyelination process, the absence of all or almost all signs of reaction except in the neuroglia tissue, and the usual absence of fat-granule cells, may be looked upon as evolving gradually to a complete sclerosis through an increasing glia hyperplasia.

B. *Through a Stage of so-called "Fat-granule Cell Myelitis."*—(a) *In the Spinal Cord* (*cf.* Figs. 47 to 52, and 53 to 56).—To trace the gradual changes by which the normal tissue is replaced by sclerotic tissue, it is convenient to refer to stages. These may be briefly described in the following terms which characterise their dominant feature:—

1. That of a commencing reaction of all the tissue components.
2. That of a glia cell proliferation and a commencing granule cell formation.
3. "Fat-granule cell myelitis."
4. That of a commencing glia fibril formation.
5. That of an advancing; and
6. That of a complete sclerosis.

Figs. 48 and 53 show the two outstanding features of the earlier process:—The development (1) of an enormous number of large glia cells with a wide zone of protoplasm and numerous branching processes

which quite mask the outline of the cell body, and (2) of rows of fat-granule cells which tend to occupy the position of the degenerated myelin tubes. Figs. 49 to 52 and 54 to 56 show (1) the gradually increasing fibril development at the expense of the glia cell protoplasm and processes; (2) the increasing diminution of the fat-granule cells in proportion to the increase in fibrils; (3) the presence of large numbers of axis cylinders in this fibrillar tissue; and (4) a gradual condensation of the walls of the blood-vessels.

The development of the large spider glia cells occurs *pari passu* with the development of the small glia nuclei, and long rows of hypertrophic cells of very varying shape may now be found, their long-branching processes passing in between the degenerating nerve fibres. The increase in the formation of fat-granule cells takes place in a very close relation to the number of the degenerating fibres, and soon the whole tissue is composed of these rows of the two cell elements, pushed aside amongst which may be found very swollen axis cylinders or granular remains of such.

The stage of "fat-granule cell myelitis" has now been reached, and the description of an "early" area applies to this area. The further evolution to an old sclerotic area consists in the further development of the fibrillar glia tissue, and here it is easily recognised how the fibril formation tends to take place in a longitudinal direction. When the tubular rows of fat-granular cells are fully formed, the glia cell processes twine around them and, when the fibril formation commences, the fibrils not only form between the rows, but pass in transversely or obliquely between groups of three, four, or more granule cells, thus forming interlacing bundles of fibrils. In the elongated meshes formed by this interlacing there would seem to be a gradual compression of the granule cells, for many seem to undergo a gradual breaking up *in situ*. The nuclei become hyperchromatic and crenated, and the definition of the cell membrane is lost, till finally only nuclei are left with a few traces of vacuolated protoplasm around them. These nuclei remain long in these glia meshes, and add to the nuclear abundance of the areas at this time. With the gradual disappearance of the fat-granule cells the interlacing bundles of fibrils gradually become more parallel: the glia cell nuclei diminish in volume as their protoplasm is differentiated into fibres; the axis cylinders, which have survived the early swelling, are now more defined, though many are still swollen and weakly stained; and, finally, the numerous vessels in the fibrillar tissue, which up till now have had their sheaths widened and filled with cell elements, gradually undergo the regressive changes already described.

The stage of advancing and complete sclerosis is one which seems to occupy a long time. It is a gradual increase of the fibrils which seem able to become more abundant, possibly through fibrillation, even

PLATE I.

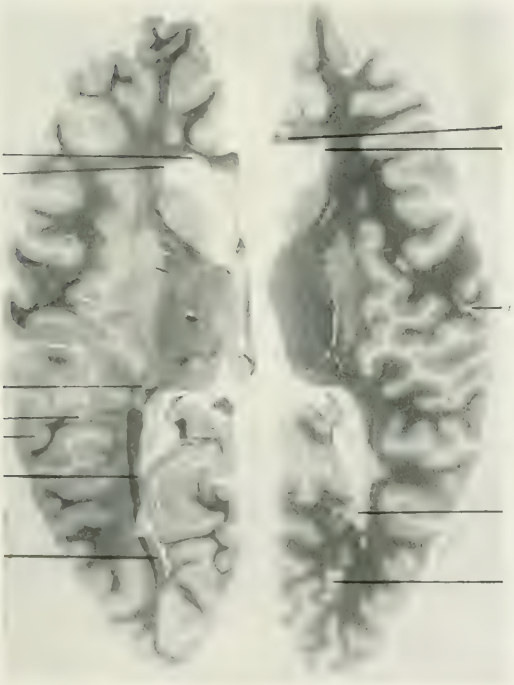


Fig. 1. Fig. 2.

PLATE II

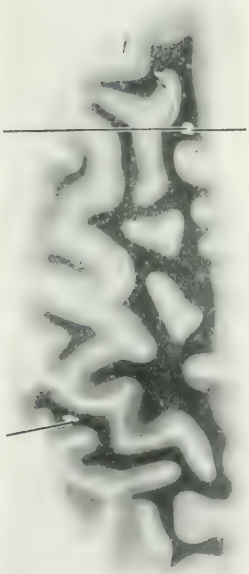


Fig. 3

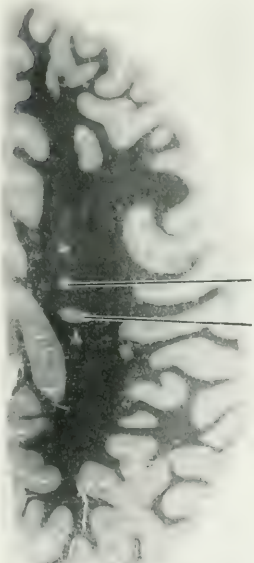
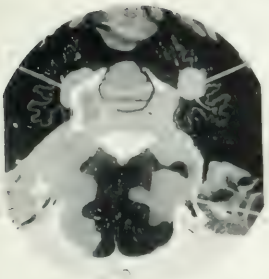


Fig. 4

PLATE III



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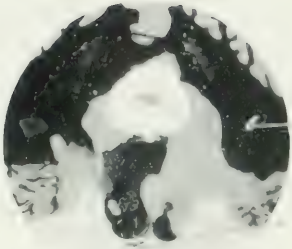

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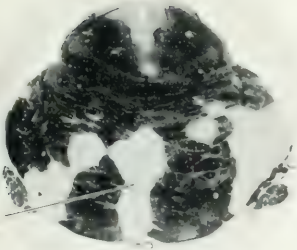

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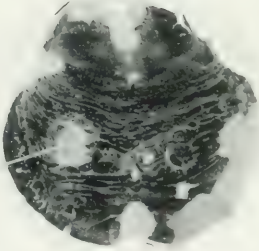

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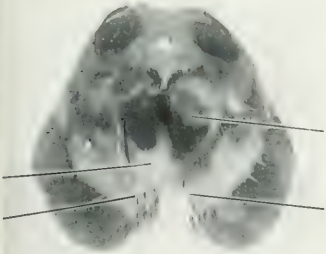

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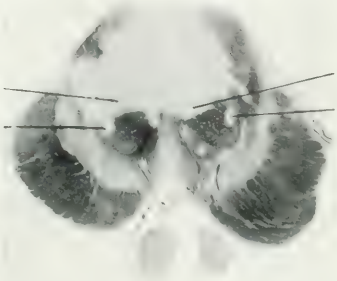
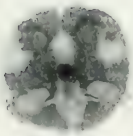


Fig. 10.

PLATE IV.



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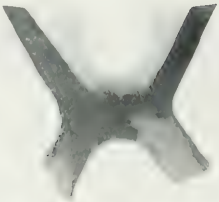

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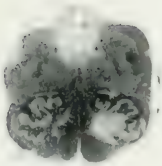


Fig. 12.

Fig. 15.

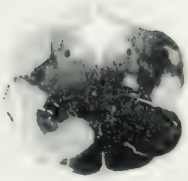


Fig. 13.

Fig. 16.

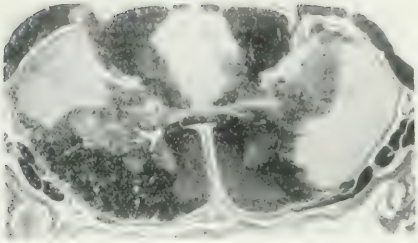
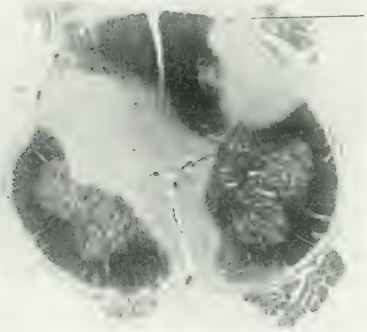


Fig. 17



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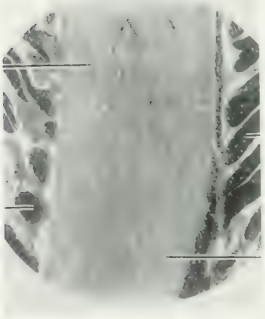


Fig. 19

PLATE VI.

Fig. 20. C_1.

Fig. 25. D_1.

Fig. 30. L_1.

Fig. 21. C_2.

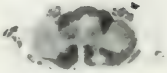


Fig. 26. D_{11}.

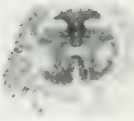


Fig. 31. L_{11}.

Fig. 22. C_3.

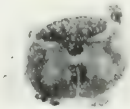
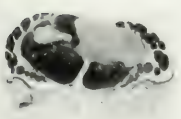


Fig. 27. L_2.

Fig. 32. S_1.



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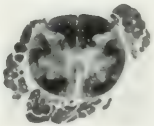
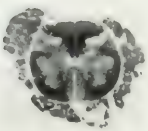


Fig. 28. L_3.

Fig. 33. S_2.

Fig. 24. D_1.



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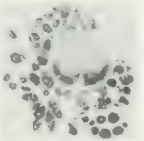

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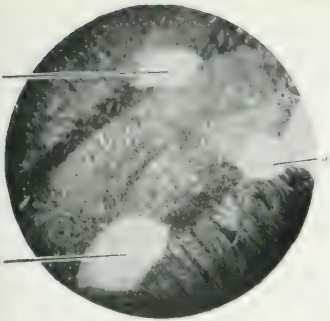

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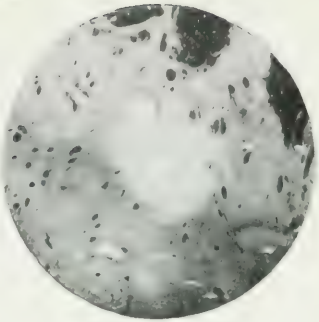

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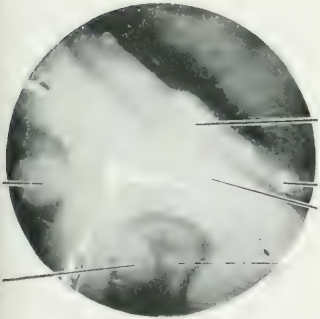

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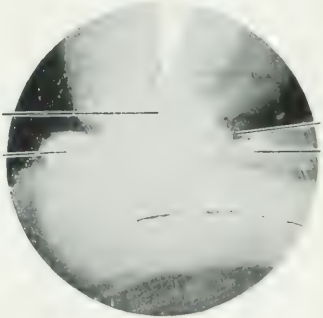
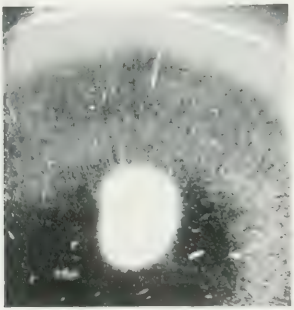


Fig. 38.

Fig. 39.



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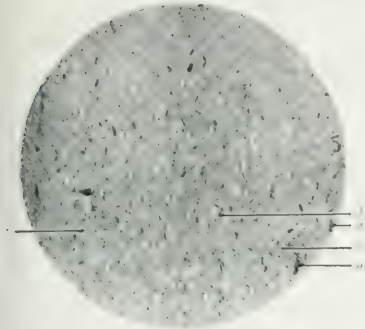

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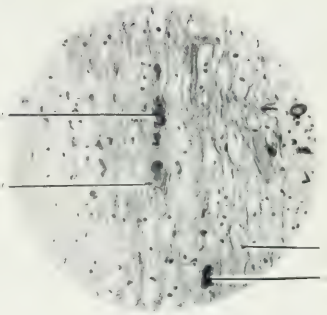

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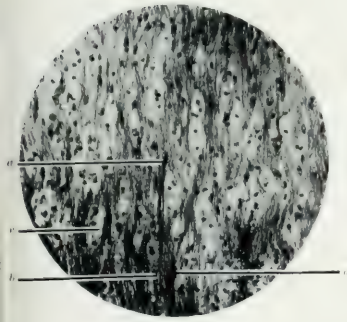

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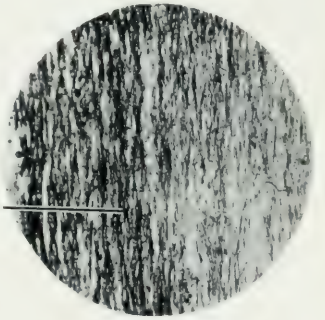

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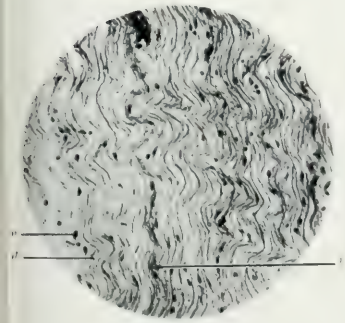

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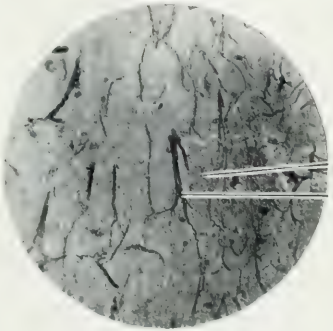
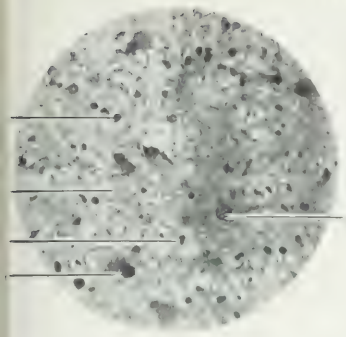


Fig. 52.

PLATE X



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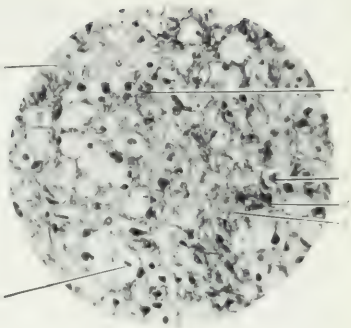

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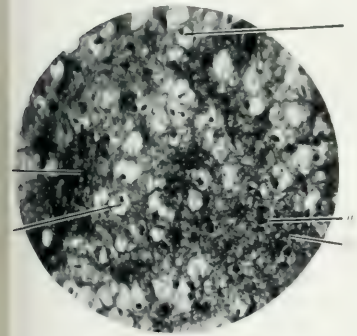

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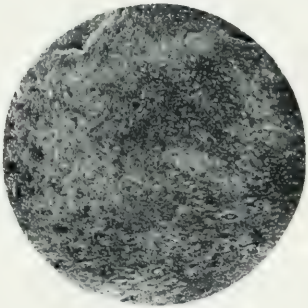

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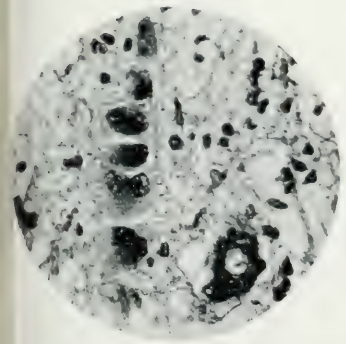

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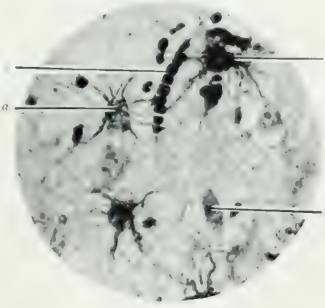

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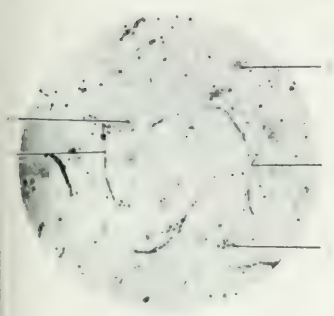

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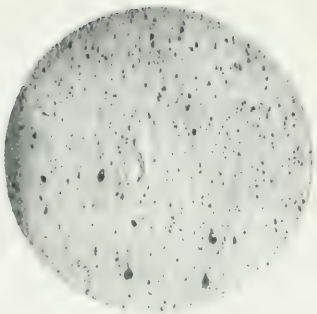

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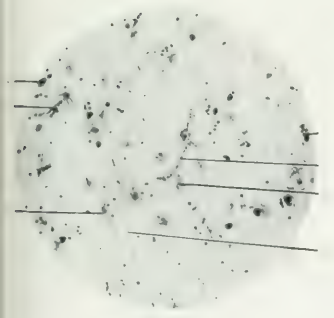

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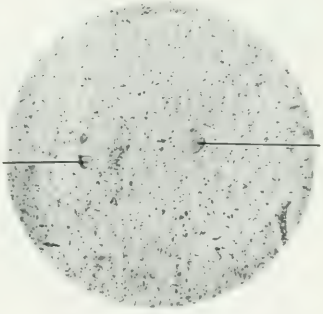

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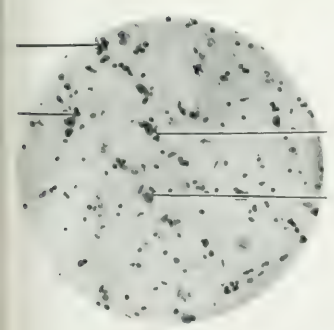

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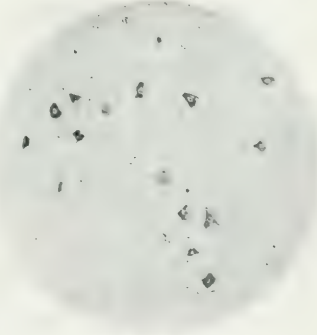

Fig. 64.

PLATE XII.

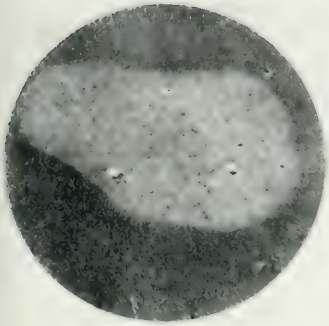

Fig. 65.

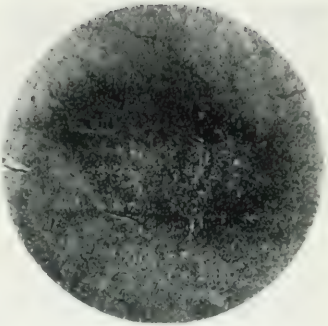

Fig. 66.

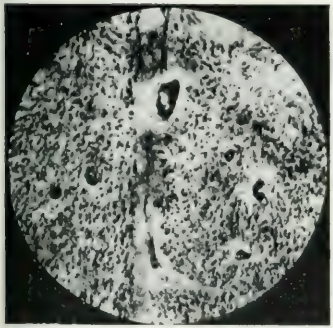

Fig. 67.

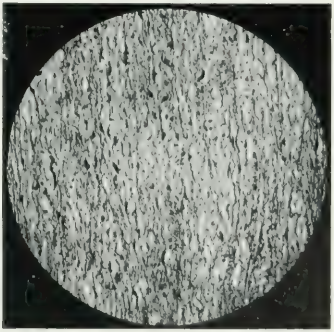

Fig. 68.

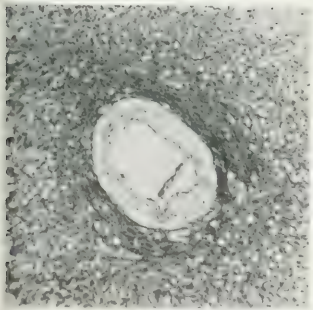

Fig. 69.

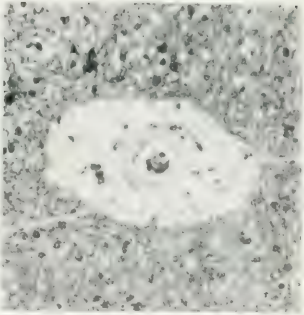

Fig. 70.

after their emancipation from the cell protoplasm. It is undoubtedly the large, pathological spider cells that produce the fibrils, and then in great part the nuclei perish. To such a disappearance and degeneration is to be traced the comparative nuclear paucity of the old sclerotic areas.

Weigert sections of such an area, during its evolution, show the successive changes of diffuse swelling of the myelin sheath, gradual loss of staining, and, finally, a negative picture with at first numerous fragments and granules, retaining the hæmatoxylin stain, scattered throughout the tissue. Marchi sections (Fig. 43) and frozen sections, stained with Scharlach R., reveal very beautifully the gradual changes in a degenerating nerve fibre —the presence of fine granules and globules or small and large irregular particles. Those changes may take place in a portion of a nerve fibre which in its subsequent course stains normally and then again shows degenerative changes.

(b) *Cerebral Area.*—The changes which occur during the evolution of an area in the central white matter show no essential difference from those just described. The nerve fibres, instead of running parallel in one direction, here run in every possible direction: when, in consequence of degeneration of the myelin sheath, or of both myelin sheath and axis cylinder, their place is taken by a dense fibrillar tissue which more or less follows the varying directions of the lost fibres, the resultant sclerosis may be described as an inextricable tangle, in which the finest meshes are invisible under low power. It would seem that it is the glia cells, which normally lie within and close to the perivascular glia sheath, which first enlarge and proliferate, and several of their broad processes end in the outer wall of the dilated vessels. With the onset of fibril formation the walls of the blood-vessels both within the area and leading from it are densely packed with fat-granule cells, amongst which may be found other nucleated elements, the result of the reaction of the adventitia to the cells in its lymph spaces. It is areas in such a stage of development that have given the justification for regarding the process as developing on the basis of an acute myelitis, for the cell infiltration of the vessel walls and the presence of fat-granule cells in such abundance in the tissue, gives all the appearance of a softened and infiltrated area. At an earlier stage, we have seen that the blood-vessel changes are not constant and that only on the onset of the removal of the fat-granule cells in the lymphatics is there any definite cell infiltration.

In the transition zone of such an area we have a gradual but slower evolution of the same changes: the sclerosis, however, never reaches the stage of a final dense meshwork, and at successive stages are found degenerating myelin fibres with hyperplastic glia cells and fat-granule cells both in the tissue and in the vessel walls—signs of an advancing process which frequently remains long after the sclerosis in the area is complete.

4. *Other Types of Areas.*—So far the histological description has been confined to areas which may be looked upon as typical of a late and of a recent process respectively, and to the evolution of a sclerotic area. As different stages occur in the same case, and as the process by no means always follows the same uniform course of development, it will be evident that the histological picture met with is a very varied one. Yet, before a comprehensive view of the complete process can be obtained, it is necessary to add to this complicated picture a brief description of other areas—areas so frequently met with as to constitute types almost as distinct as those already taken of an old and a recent process.

(a) *Areolar Areas.*—These may be related simply to an arrest of the fibre formation before a dense sclerosis had taken place: the transition zone around old areas would seem to justify this explanation. Others must be traced in all probability simply to an acute and rapid degeneration of the nerve elements, with a distension of the original glia meshes, before fibril formation had occurred.

(b) *Perivascular Sieve-like Areas.*—Non-myelinated areas are frequently found, especially in the brain, in which large open spaces are met with around all the vessels present within the affected areas. The vessels thus affected lie together in groups and give the tissue a sieve-like character which, when advanced, has been termed *l'état criblé*. The impression is received that it is the branches of one vessel stem that are concerned in this change.

(c) *Shadow Sclerosis.*—Extensive patches which show a weak staining of the myelin are frequently found between individual non-myelinated areas. The fibre bundles are distinctly perceptible and yet scarcely stained. Such areas may involve the whole transverse section of the cord or the longitudinal section over several segments. Alzheimer looks upon this deficient staining as a condition of the myelin sheath antecedent to degeneration, and Marchi sections sometimes give a very extensive early degenerative process with which an equally extensive commencing glia proliferation may be associated. The process may, however, be looked upon as a slumbering one and indicate a simple atrophy which results in the progressive reduction of the volume of the sheath without affecting the remaining myelin.

It may finally be noted that in Marchi sections of the cord there was frequently found extending, outside the sclerosed area, uniformly over the whole transverse section an early myelin-sheath degeneration. This could be noted even in the nerve roots, and must probably be traced to the septic fever from which the patient suffered.

(3) Histological Characteristics in Special Situations.

1. *White Matter.*—This has already been fully considered, as the areas described in the previous sections were all in the white matter of the brain and cord.

2. *Grey Matter.*—(a) *Central Grey Matter.*—Where the sclerotic process affects the grey matter of the spinal cord and the corresponding nuclei of grey matter in the medulla oblongata and pons, we find that the changes in their evolution correspond very closely to those in white matter in which the nerve fibres form a reticulum.

The myelin network quickly disappears, and in its place we get a thickened glia reticulum with large meshes and numerous hypertrophied glia cells. These spider cells, with relatively numerous processes, varying in length and thickness, give rise to a fibril formation analogous to that already described—the resulting meshwork being very close. The formation of fat-granule cells is always much less marked than in the white matter; their size is smaller and the structure of their granules more delicate. This corresponds to the lessened quantity of myelinated fibres in the grey matter, but the process of their formation, the absorption of the degenerated myelin, their presence in the glia meshes, and their gradual removal in the lymphatic spaces of the vessels, is quite similar to that elsewhere.

It is frequently stated that sclerotic areas are never found solely in the grey matter, but that these are always extensions from sclerosis of the white columns. In serial sections, however, it could be proved that small sclerotic areas may be confined, throughout their whole longitudinal extent, to the grey matter. An affection of the myelin reticulum around and between one or other group of cells (Figs. 28 and 33) has frequently been the sole trace of demyelination in many sections of the lumbar cord. A pericentral sclerosis (Figs. 30 to 33), limited at first entirely to the commissures, is often the starting point of an extension into each anterior and lateral horn. Undoubtedly, however, the affection of the anterior horn of grey matter of the cord starts very frequently from the extension inwards of an area of sclerosis situated at the margin between white and grey matter.

(b) *Cortical Grey Matter* (Figs. 37 to 40). Numerous minute demyelinated areas were found in all the layers of the cortex, while large numbers passed over from the subcortical white matter into the cortical layers. Early writers asserted that the grey matter formed a barrier to the extension of the process, and numerous recent writers emphasise the complete absence of a glial sclerosis as the essential characteristic of a cortical area. The examination of very numerous areas has led to the conclusion that a glia fibril formation in the layers of the cortex is proportionate in its development to the normal glia fibril content of the layers, and that a change in the glial cells and fine glia reticulum of the cortex is very closely related to the loss of its myelin fibre content.

In considering the structure of a combined subcortical and cortical area it is sufficient to emphasise the changes related to the glia cell, glia fibril, and fat-granule cell development, for the myelin sheath

and axis cylinder changes do not differ from those in other areas (Figs. 59 to 61). In the transition zone the deepest layer of the cortex is no longer recognisable: in the layer of the deep pyramids and Betz cells both glia cell proliferation and fat-granule cell formation are still very marked; from the granular layer upwards, however, the intensity of the cell reaction is much less. Nests of small round cells are found around nuclear remains of atrophic ganglion cells and around others, and around the capillaries is found a fine fat-granule cell formation in the satellite cells, together with a very delicate glia fibril formation. In the marginal zone there is again a marked glia cell and fibril formation.

If the evolution of such an area be followed, it is found that in the white matter and deepest layers the process often follows the lines one would expect from the presence of the large, protoplasmic, potential fibril-forming glia cells. This sclerosis extends to involve the deepest layer of the cortex. Above this, in the layer of deep pyramids and Betz cells, the glia cell nuclei are left as the nodal points from which radiate a glia fibril formation which is insufficient to cause sclerosis; the fibrils, however, merge into a very delicate reticulum, which may be the syncytium claimed by Held to form the groundwork of the cortex. In the superficial layers of the cortex the long, uniform delicate processes of the glia cells also unite with this reticulum, and in the surface layer the proliferated glia cells form fibrils which extend downwards—also to merge with the syncytial reticulum.

When an area is confined to the cortex, the changes are, as a rule, not nearly so marked, especially those in the deepest layers. The demyelination may reach from the surface of the convolution to varying depths, or it may lie wholly within the cortex and cut through a portion of the radiation, or simply affect Baillarger's stripe. The glia cells in the layer of the deep pyramids show a marked proliferation and fibril formation, which, however, is again insufficient to cause sclerosis. In the layer of stellate cells also there are the same changes with a more marked disappearance of the ganglion cell bodies, leaving only nuclei surrounded here and there by nests of cells.

3. *Periventricular Sclerosis.* — This was the dominant feature in several of the cases. This special localisation is of great interest in relation to the pathogenesis of the disease, and led the late Dr. Bruce to raise the question of the toxicity of the cerebro-spinal fluid.

Horizontal sections through the hemispheres at various levels (Figs. 1, 2) showed that the occipital horn of the lateral ventricle was surrounded by a hood of changed tissue, macroscopically greyish-white and gelatinous or of a whitish yellow colour and softer consistence. From the point of this hood the sclerosis is prolonged in a series of small elongated or rounded areas towards the posterior extremity of the occipital lobe, involving the optic radiations, the inferior longitudinal

fasciculus, and the tapetum at several points, and in some cases involving the medullary rays and cortex of the convolutions of the calcarine fissure. The sclerosis of the anterior horns is less marked, but is also prolonged in the direction of the frontal lobe, and laterally involves the lenticular nucleus and the cortico pontine fibres. Between these two horns the thickness of the sclerotic tissue varies greatly at individual levels, and may show well-marked processes extending into the grey nuclei and adjoining white matter. The fornix and the corpus callosum, on the ventricular surface both of the splenium and genu, may also be involved.

Horizontal sections near the roof of the ventricle usually showed an involvement of the entire surface extent of the ventricular walls, with well-marked pouches passing inwards into the corpus callosum on the one side and the central white matter on the other and also passing upwards.

Sagittal sections through the temporo-sphenoidal lobe gave a very instructive picture of the extension outwards of the ring of peri-ventricular sclerosis into each medullary ray and of the very frequent involvement of the white matter of the hippocampal convolution, of the fimbria, and of the gyrus dentatus.

Around the aqueduct of Sylvius and the floor of the fourth ventricle and its lateral angles (*cf.* Figs. 5 to 10), the degree of sclerosis is often very marked. The involvement of the cranial nuclei in this extension is described later.

The ventricles in most of the cases were not dilated; their walls also were smooth and presented neither granulations nor glandular indentations. The histological structure of these demyelinated areas need not be entered into in any detail. When the immediately subependymal tissue had reached the stage of dense sclerosis it presented an extremely close fibrillar web—the fibrils mostly parallel or oriented in all directions. Before the onset of this advanced sclerosis numerous dilated vessels twined amongst rarefied and elongated glia meshes.

4. *Changes in Nerve Roots and Meninges.*—(a) *Optic Nerves* (*cf.* Figs. 14 to 16). The optic nerves, chiasma, and anterior portion of the tracts, investigated in seven of the cases, showed marked involvement; the chiasma was the site most frequently affected. In one case the optic tract on both sides was cut in celloidin sections from the chiasma to the corpora geniculata interna, and both showed a very characteristic discontinuous degeneration; the degeneration in the chiasma passed into both optic tracts and then ceased, to begin again at a point midway in its course. In a second case completely gelatinous and demyelinated optic chiasma and nerves showed an almost complete preservation of the axis cylinder. The histological characteristics are related to the presence of both glia and connective tissue interstitial elements, for

both are affected; in the advancing sclerosis the glia fibril formation is almost masked by the great connective tissue increase, which the very numerous blood-vessels of the septa had shared.

Sclerotic areas in these regions are amongst the most constant appearances in disseminated sclerosis, and may be related to the early eye changes found clinically.

(b) *Cranial and Spinal Nerve Roots.*—Numerous lesions were found in the glious portion of the nerve roots (Fig. 18*a*); the extent of this demyelination and neuroglial involvement varied greatly, and it was looked upon as proportionate in extent and form to the varying degree in which the glia normally passes into the nerve roots.

(c) *Changes in the Meninges.*—The cerebral and spinal meninges were found almost normal in uncomplicated cases; meningeal changes when present were diffuse and in no way confined to the meninges overlying areas of sclerosis. Borst, in four cases examined by him, found adhesions and thickenings: the significance ascribed by him to these changes is of great importance, in relation to the pathogenesis of the disease.

(4) Changes in the Individual Tissue Elements.

1. *Nerve Fibres.*—(a) *Medullated Sheath.*—The change in the myelin sheath must be looked upon as the most constant, the most uniform, and in many cases the primary one. In the actual sclerotic areas the medullated sheath had entirely perished, but in recent areas and at the advancing peripheral zone of older areas the changes present themselves in very various forms according to the stages of the degenerative process, and according to its greater or lesser intensity.

In longitudinal sections of the cord it may be seen that the degeneration may affect the nerve fibres "discontinuously." This is specially easily recognised in Marchi sections, counter-stained with safranin, in which the retained axis cylinder is seen surrounded by a pink-stained zone, which becomes gradually interrupted by a portion of the fibre in which granular disintegration has commenced. The axis cylinder within this affected portion may be swollen and homogeneous, but can be recognised till a late stage of the degeneration. Thus this form of degeneration can be distinguished from secondary degeneration by its limitation, by its late affection of the axis cylinder, and finally, by the character of its disintegration into granules and globules instead of at once into coarse balls and fragments which fill the whole myelin sheath. The fat-granule cells, which are found in such large numbers, could only in rare instances be found to contain large fragments of myelin. It seemed rather as if a gradual absorption of dissolved substances had taken place, and that these within the cell had become transformed into fine fat-like granules.

(b) *Axis Cylinder.*—The comparative persistence of the axis cylinders

in the sclerotic area has long been regarded as one of the essential characteristics of disseminated sclerosis. Charcot himself, from the apparent disproportion between the symptoms and the anatomical lesions, argued for their persistence. The earlier observers undoubtedly, with diffuse stains, frequently mistook the longitudinal glia fibrils, which run parallel to the degenerated nerve fibres, for true axis cylinders, yet modern specific staining methods have proved that Charcot's conception was justified. Yet this persistence is only a relative one, and attention has already been drawn to the numerous alterations which axis cylinders undergo. The most frequent change is that of a slight homogeneous swelling, and this may be found even in old sclerotic areas, in which, as a rule, the axis cylinder becomes attenuated, and with diffuse stains is only with difficulty distinguished from the coarser fibrils. The changes in the axis cylinders are much more easily recognised in longitudinal sections of the nerve fibres.

In early areas degenerative changes set in with a moniliform appearance, which may later take the form of spindle-like enlargements of various size, and these may finally lead to disintegration, with the formation of homogeneous clumps and granules. This qualitative change may affect a large number of the axis cylinders in an affected area, or most of the fibres may show spindle-shaped swelling and only a few go on to the stage of disintegration. It is probable that the fibres which survive the swelling may persist into the stage of sclerosis as homogeneous condensed elements which, as the sclerosis becomes denser, are compressed into thin, even spiral threads.

The first effect of the "toxine" may thus be a lecitholysis; when this is carried further or is more intense there is a marked axolysis with the formation of fine granules. But a change short of this—the demyelination and the swelling of the axis cylinder, which is an indication of its sensitiveness and a proof of it sharing in the changes in the myelin sheath—may be present.

2. *Nerve Cells.* The relative resistance ascribed to the axis cylinders must also be extended to the nerve cells, both in the cord and brain. The ganglion cells in the glia reticulum seem to remain long preserved, and in this investigation changes which could be distinctly traced to the sclerotic process were found only when an advanced degree of sclerosis had been reached. The cells undoubtedly remain long with their normal form and minute structure preserved. The processes first lose their structure and, as the sclerosis becomes denser, the cell bodies share in this change. In the later stages they show all the possible changes which are traceable to a slow simple atrophy from loss of function (Fig. 64) or from compression on the part of the developing cells and fibrils of the interstitial tissue. Diffusely extensive changes in the ganglion cells should probably be referred to somatic general disturbances which accompany the disease, *e.g.* the fever, anæmia, exhaustion,

or direct septic absorption from bed-sores or a pyelitis, and can thus not be looked upon as specific to disseminated sclerosis. In numerous affected segments which microscopically showed complete demyelination many of the ganglion cells showed no changes — completely normal cells being found alongside those with marked chromatolysis or atrophy.

As the glia reticulum becomes denser and encroaches more and more on the ganglion cell processes and cell body there takes place a gradual atrophy of the cell. For a long time an atrophic, rounded, or oval cell, with no processes and no chromatophile granules and usually without nucleus, may be recognised. But gradually this faintly or diffusely staining body can no longer be recognised as a cell, and this dense tissue consists of the deeply-staining glia nuclei and the abundant delicate fibrils.

The cells in the different cranial nuclei undergo changes which are closely analogous to those described above.

The cells in the cortex, in a demyelineated area, when involved, are much more uniformly so than those of the cord. The large pyramidal cells are never normal (Fig. 62), but show chromatolysis and have their dendrites thickened, or weakly stained, or absent, and the cell contour is rounded or pear-shaped. These changes involve not only the demyelinated area but also the adjoining stretches of the cortex to a varying extent. But the most characteristic appearance in a cortical area, and that which enables it, under low power, to be readily recognised, is the nuclear increase around the ganglion cells. The majority of the ganglion cells, in the lower layers of the cortex and in the layer of the large pyramids, are surrounded by small nests of cells (Fig. 61), from five to ten in number. These with diffuse stains have a small deeply-staining nucleus with little protoplasm, and cause, according to their number and arrangement, more or less deformity and atrophy of the ganglion cell, but I have never seen the penetration of such cells into the ganglion cell body. They have probably arisen from the pre-existing satellite cells, and their proliferation is taken as an indication of the inter-relation between ganglion cell and its satellite cells. Marinesco thinks that in health the former secrete substances which exercise a controlling influence on the size and development of the latter: if any noxious or toxic agent affects the former, this controlling secretion is diminished in quantity and quality, and the neuroglia cells react accordingly by increasing in size and numbers. This change is again not limited to the demyelinated areas, but passes over to the adjoining stretches of the cortex.

Comparatively little change was found in the cells of the posterior root ganglia. Those that were present indicated a general reaction, probably in no way related to the sclerotic process in the cord.

3. *Neuroglia.*—The changes in the neuroglia represent, in the

opinion of numerous observers, the dominant and essential histological feature of disseminated sclerosis. Whilst the changes in the nerve fibres show little variation in the individual cases, the glia components show wide differences. In an early area we have seen that the most important and characteristic finding is the enormous number of large cell elements which, in their further evolution, are transformed into glia fibrils and glia nuclei. In an actual sclerotic area we have a dense glia fibril mass, apparently without spaces. The histological characters of the glia, therefore, depend on the length and the intensity of the process. In the final result the name "sclerosis" is justified by the amount of neuroglia present in the areas, and Weigert has stated that the glia hyperplasia is greater here than in any other form of sclerosis. The neuroglia, though not from a histogenetic, yet from a morphological and biological standpoint, may be looked upon as a true fibrous connective tissue. It is thus of great significance that the glia begins to proliferate when component parts of the specific nervous tissue are destroyed, even where any stimulus capable of causing primary proliferation is absent. The classical example of this is the secondary degeneration of the white substance, where the secondary glia proliferation sets in probably as a reaction to the irritation caused by the products of degeneration. At the same time it may happen that one and the same "noxa" destroys the specific nerve tissue, and in an equally primary manner causes the glia to proliferate. Again, it is possible that we may have isolated primary proliferative processes in the glia—which in their turn cause secondary degeneration of nerve cells and fibres. Storch has pointed out that in chronic diseases, in which the plan of the nerve tissue remains unchanged, the newly-formed glia fibrils show exactly the same arrangement as the original fibres, whereas in cases of acute destruction of tissue this regularity does not hold good. He, therefore, distinguishes between an isomorphous and a reparatory sclerosis. The glia proliferation may, therefore, be merely a substitution process, or an inflammatory process, or, more rarely, a primary glia proliferation.

It is impossible to discuss here the question of the spatial relation of the glia fibres to the glia cell protoplasm, or to indicate the stages in the elaboration of the glia fibrils (*cf.* Figs. 58 to 60). Glia cells that have produced fibrils undergo slow and gradually regressive changes: almost the whole of the protoplasm seems used up and shrivels into an irregular border around the deeply staining nucleus. In later stages, too, the nuclei may disappear, and thus account for the comparatively few nuclei found as a rule in actual sclerotic areas.

In addition to fibril formation the glia cells share in the function of phagocytosis, for one portion of the proliferated glia cells, both in the white and grey matter, probably forms the first fat-granule cells.

The increasing zone of protoplasm around the nucleus becomes gradually laden with granules which often outline the cell body and its processes—at first spindle-shaped, then star-shaped, and gradually becoming rounder till the cells are set free in the glia reticulum. These glious fat-granule cells have a lattice or "gitter" structure in their protoplasm, in preparations in which the fat-granules have been dissolved out.

4. *Blood-vessels; Lymphatics; Cell Elements in Vessel Walls.*—(a) *Blood-vessels.*—The disposition of the areas in relation to the blood-vessels has led to the supposition that these play an important *rôle* in the genesis of the areas. This topographical relation is frequently obvious even microscopically, especially in areas in the cerebral white matter, and numerous illustrations point to its microscopic proof. This dependence on the vessels is brought to light only where the areas are isolated; in later stages, through coalescence, the original relationship is no longer recognisable. A parallelism between the sclerosis and the area of distribution of a vessel would argue that the development of the process depends upon the condition of the vessel or of the "noxa" circulating within it. To the alterations in the vessel walls in sclerotic areas numerous writers have, therefore, ascribed an essential significance, while others have regarded them as accessory and subordinate. Variations in the findings may be explained by the different stages in the development of the sclerotic process, and probably none of the histological changes have been so variously interpreted as those related to the vessels.

The sequence of the changes in the blood-vessels seems to me to be the following: at an early stage the blood-vessels in an involved area become dilated and engorged with blood. It is not a question so much of one central vessel as of all the branches of one small vessel, and even the finest capillaries are recognised; the vessels are in no way altered, and have no nuclear increase either in the intima or adventitia. At the stage of abundant fat-granule cell formation, when these cell elements are passing into the lymph spaces of the adventitia, every vessel in the affected zone is mapped out by a ring or rings of such cells. The smallest capillaries show a single row of cells, often with an outer limiting membrane. If such an area, with fat-granule cells crowding the vessel sheaths and tissue spaces, be looked at with low power, the impression is given of a softened area with cell-infiltrated walls. The possibility that areas at such a stage of development have been taken as illustrating cell infiltrated areas may explain the great significance that has been ascribed to the vessels and to the inflammatory character of the process. This is still more evident at a slightly later stage, when there is a proliferative reaction of the cell elements of the adventitia to the presence of these fat-granule cells in the lymphatic spaces. As the resorption processes advance there is a

gradual removal of the fat granule cell element in the adventitial spaces, but the cell body of many of these cells has gradually disappeared *in situ*, leaving a deeply stained, cremated nucleus, and during this stage we get a further nuclear element added to the nuclear increase in the vessel wall—this is the small, round, lymphocyte-like cell common to all chronic processes. These, with the elements distinctly recognisable as proliferated from the endothelium of the adventitial spaces and from the adventitial connective-tissue cells, together with a few remaining fat granule cells or their nuclei, form the cellular content of the vessel walls at this stage. As the sclerosis advances the dissociated fibres of the adventitia gradually come together, and proportionate to this is a gradual diminution in the contained cell elements. At a still later stage the sclerosis affects all the parts of the vessel wall, and the dissociated fibrils form a fused, homogeneous, almost hyaline, layer. As the vessel becomes hyaline the elastica becomes broken up and lost, so that nothing can be recognised of specific muscle, connective tissue, or elastic elements.

In the majority of the sclerotic areas all the vessels are condensed arteries, veins, and capillaries—and it is not possible to draw any distinction between arteries and veins, arterioles and venules. Similar condensation of the vessels is found in all chronic sclerotic conditions in the nervous tissues, and Lugaro has pointed out that this is in keeping with the small claims of their nutritive function, for the sclerotic tissue requires less nourishing material.

(b) *Lymphatics.*—The increasing recognition of the importance of the lymphatic circulation in the central nervous system has led to numerous researches upon the precise nature of the lymph paths and the direction of the flow of the lymph within them. It cannot be said, however, that any exact knowledge on either of these points has yet been revealed. The present section of this paper is confined to the indications of changes in the lymphatic vessel system in disseminated sclerosis. The lymph apparatus shares in the changes in the blood vessels, and Borst has strongly urged the view that a disturbance in the lymphatic circulation is the essential factor in the process underlying disseminated sclerosis. He points out that the factors which bring about this disturbed circulation are alterations in the vessel walls and in the meninges. Even slight affections of the former involve changes in the transudation of the lymph and lead to pathological exudations, and even slight adhesion of the pia to the marginal glia zone would involve occlusion of the epispinal or epicerebral spaces, an occlusion which would be extended to the perivascular spaces at the periphery, and therefore cause a hindrance to the outflow of the lymph by these channels. In two recent papers the late Dr. Bruce and I indicated our agreement with Nissl's contention that the adventitial lymph spaces of the blood-vessels, which open

into the spaces of the inner layers of the pia, are the only true lymph spaces of the central nervous system. It has seemed, therefore, that this assumption of epispinal, epicerebral, and perivascular spaces is an error which invalidates greatly Borst's views, and causes him to overestimate the part played by these adhesive changes. The fundamental importance of an increased transudation of toxic lymph cannot be overestimated, but the evidence of a hyperlymphosis is not by any means clear except in isolated cases, and its explanation when present cannot always be traced to be secondary to a closure of adventitial spaces.

In the course of this study attention has been directed to the fact that the adventitial lymph spaces showed in the early stages only a slight decree of dilatation; that, later, they were distended by the presence of fat-granule cells and remained dilated and filled with cell elements of various kinds till a late stage of sclerosis, when they were closed in the general condensation of the vessel wall. There was no question, therefore, of a primary proliferation such as might be induced by a toxic lymph flowing in them.

Very numerous perivascular sieve-like areas were found, but these were frequently related to normally myelinated tissue. There were also found indications of a lymph congestion which extended over the whole transverse section of the cord and which found expression in dilated adventitial lymph spaces, distended glia meshes, and a swelling of the contained myelin sheath and axis cylinder. In uncomplicated cases only occasional and insignificant variations in the soft meninges were present, yet it must be admitted that changes, if minute, might readily escape observation.

(c) *Cell Elements in the Vessel Walls.*—The statement is frequently made that infiltrations are met with in recent areas, and such cell infiltrations of the adventitial sheath are considered characteristic of the inflammatory process in the nervous tissue. In numerous references to the cell elements of the adventitia an endeavour has been made to show that the first cell infiltration is one associated with resorption processes; that this is followed by a cell proliferation in the adventitia, which is probably a secondary reaction process to the presence of the first cells in the lymph spaces; and that, finally, we have a more or less marked degree of cell infiltration which is characteristic of a chronic inflammatory process in any tissue.

(5) Other Histological Features.

1. *Form and Summary.*—Rossolimo first drew attention to the dependence of the sclerosed areas upon the topographical distribution of the blood-vessels. As the causal agent spreads itself by the blood-vessel or by the lymph channels accompanying the vessels, it was asserted that the areas assume certain well-defined forms. These basal forms —

wedge shaped and round or oval—are in agreement with Kalyi's experimental observations on the arterial distribution in the cord; the lateral vessel passes transversely into the cord substance and its first branches are given off transversely, but the transitional vessels run upwards and downwards in the long axis of the cord. The area of distribution, therefore, of the transverse branches is wedge-shaped and that of the perpendicular branches is an elongated oval. Such vessels have been looked upon as end arteries, but there is no doubt that there is a considerable amount of overlapping. The blood supply likewise of the groups of ganglion cells is not from one individual branch of the commissural vessels but from several—the sulco-commissural arteries form in the substance of the horn a rich and intimate network which holds the cell groups enweaved in its meshes. In the course of this study several small areas have been followed up serially throughout their whole extent, and I have come to the conviction that the changes appear within, but do not coincide with, the area of distribution of an artery, and that it must be extremely difficult to determine the territory of an artery.

In the cord the prevailing form of subpial areas is wedge-shaped, but when a large portion of the peripheral part of the cord is affected, perhaps through fusion of contiguous lateral areas, the definition is irregular. Within the white substance of the cord the areas are frequently round or oval, with the long diameter in frontal or sagittal section. Isolated areas may occur in the grey matter, especially of the anterior horns, and such areas may involve one group of nerve cells or may map out the whole of one horn as a demyelinated area; the most frequent involvement, however, is a pericentral sclerosis, which may be very marked at almost every level of the cord. The longitudinal extent of areas within the white matter is very various and sometimes reaches over several segments. In the brain stem the same two primary forms are present with numerous variations. The cranial nuclei shared in the general extension from the floor of the fourth ventricle; this extension passed laterally from the angles of the ventricle inwards to involve the structures which at various levels are found in this region. The lateral extension to the roof of the fourth ventricle involved the white matter of the cerebellum, the hilum of the dentate nucleus, the nuclei of the roof and often the whole of the foliæ forming the vermis and nodules (*cf.* Figs. 5-10).

In the medullary rays of the convolutions numerous submiliary foci were present, with the long diameter usually parallel to the course of the nerve fibres. In the cerebral cortex the variation in the form of the areas is even more marked than elsewhere. These may be grouped into three divisions, according to whether the demyelination (1) spreads from white matter into grey and is arrested within the radiations of the nerve fibres, or (2) is wholly within the radiations of the nerve

fibres, or (3) is subpial. The close disposition of the numerous areas in certain convolutions gave place often to a coalescence, with a complete demyelination of cortical tissue, extending over the whole of one convolution and sometimes round the cups of the adjoining convolutions.

The lesions are distributed over the whole central nervous system, and, as they do not coincide with the area of distribution of a vessel, their form can only in a modified way be related to the two basal types. Sites of predilection are probably associated with the terminal ramifications of end-arteries, the points where vessels break up, and with areas where much glia is normally present. The frequent marked symmetry of the areas may also be related to these circumstances.

2. *Secondary Degeneration.*—The absence of secondary degeneration has been till recently accepted as one of the cardinal characteristics of the histological picture. Charcot related this feature to the preservation of the axis cylinders in the sclerotic tissue, and it has been shown that complete demyelination of the nerve fibres at a circumscribed level occasions no secondary degeneration even in the myelin sheath, and that this follows only a considerable alteration of the axis cylinder itself.

The question of secondary degeneration is one not easily decided. It is probably at first confined to individual fibres, and, later, the difficulty of recognising it in cases where several islets of sclerotic tissue are present is increased by numerous factors, such as the presence of the diffuse changes which unite sclerotic areas or the presence of more acute processes arising in the affected columns. It may be noted that numerous and only microscopically evident areas may be found in the course of one and the same conducting tract.

3. *General Features and Distribution of Areas in Case I.*—The number of areas was very large and their distribution throughout the central nervous system was very extensive. The parts most involved were the cervical enlargement of the spinal cord, the medulla oblongata and the pons, and the periventricular tissue. The distribution of the areas in relation to the floor and walls of the fourth ventricle and to the hilum of the dentate nucleus, together with the prominent involvement of numerous folia in the cerebellum of both flocculi and of the peduncles (*cf.* Figs. 5, 6) was very striking. Both optic nerves were extensively demyelinated and nearly all the cranial nerve roots entered into sclerotic tissue. The nuclei of the sixth nerve, the cochlear nuclei, and the nuclei of Bechterew and Deiters were all involved. The posterior horns of the lateral ventricles were surrounded by large irregular areas of sclerosis, which extended at several points through the white matter to the surface of the brain. This was well marked on the inner side of both posterior horns where the sclerosis cuts across the tapetum, the inferior longitudinal bundle, and the splenium, and reaches the surface at

the parieto-occipital fissure. On the right side this area was continuous with another which extended from the angle of the ventricle to the foot of the calcarine fissure. Numerous small areas were present in the basal ganglia (*cf.* Figs. 1, 2).

The most striking histological feature was that almost every area showed evidence of an advancing process; the impression left was that the primary affection had never died down, but had gradually extended peripherally while the central sclerosis had also extended excentrically (*cf.* Figs. 44, 45).

(6) Note on the Pathological Physiology.

There is no disease of the nervous system in which the symptoms may be more varied, in origin and in succession, than in disseminated sclerosis, a fact which can be well understood when one considers the wide range and irregular distribution of the areas of sclerosis. These, as we have seen, may occur in almost any part of the nervous system, producing peculiar symptoms and combinations of symptoms, which may be of short duration and are usually followed by periods of remission, after which a new series of symptoms may appear. Once definitely established the disease is almost invariably fatal. The great variation in the nervous symptoms has resulted in a tendency, on the one hand, for any disease of nervous origin of which the symptoms are unusual or difficult to interpret to be classified as "disseminated sclerosis," while, on the other hand, the way in which the symptoms of a case of true disseminated sclerosis may simulate other nervous diseases has often led to errors in diagnosis. On account of the long duration of the disease in most cases, and the unsatisfactory results of treatment, patients are not kept in hospital for long periods of time, and thus the number of cases which come to autopsy is not large.

The earliest symptoms of the disease are extremely variable, and depend solely upon the particular part of the nervous system affected by a patch. There is, however, a tendency for these patches to occur more frequently in certain regions, and thus certain clinical symptoms appear more commonly than others, and as a result are considered more or less pathognomonic of this condition.

Of these symptoms one of the commonest is weakness of the legs. This is met with in a very large number of cases, and is associated in most with involvement of the spinal cord. It usually is progressive, and a spastic paraplegia develops. It is often accompanied by a number of more or less well-defined sensory changes. Such symptoms may result from one or more patches

in the cervical or dorsal regions of the cord, involving the descending motor and adjacent sensory fibres. If the area involved be still lower, *e.g.* in the sacral region, then the only symptom may be a sphincter involvement. These spinal symptoms may be the only ones present, or they may be accompanied or followed by others due to involvement of the higher centres. Of these, the principal ones most commonly found are nystagmus, alteration in speech, and volitional tremor. These are largely due to want of co-ordination, and are the result of sclerosis spreading in from the ventricles.

The eye symptoms are of special importance, and are very often the first sign of the disease. They often disappear quickly or may ultimately proceed to total blindness. They are the result of patches in different places in the optic path, the optic radiations as they pass near the descending horn of the lateral ventricle being commonly involved in the periventricular sclerosis. The later and more serious symptoms, however, result from involvement of the more peripheral part of the optic path, namely, the optic tracts and chiasma. The affection of the eye muscles occurs so frequently from the close relationship of the structures innervating them to the ventricular spaces, the peri-aqueductal sclerosis found in the mid-brain involving the third nucleus in many cases. The nucleus of Deiters and other vestibular nuclei situated at the angles of the fourth ventricle are also specially liable to involvement, and there is no doubt that it is the proximity of these structures to the ventricles which accounts for their frequent involvement and the special diagnostic importance of nystagmus and visual symptoms.

The "volitional" tremor and scanning speech are also symptoms of the very greatest importance. They are both the result of defects of co-ordination. The tremor occurs only on voluntary movement, and is absent during rest. In attempting to carry out any movement the arm jerks about in an irregular manner, the tremor becoming quicker towards the end of the voluntary act. This form of involuntary movement, we know now, results from affection of the cerebello-rubro-thalamo-cortical path. Injury to this path allows involuntary movements to occur by removing the steadying influence which it normally exerts upon the Betz cells in the motor area. If this influence be removed, steady innervation of the anterior horn cells is impaired, and the more the pyramidal path is innervated, the more obvious does the tremor become. Involvement of the cerebello-rubro-thalamo-cortical path

may occur, thus, at many different levels, and any of these lesions may result in this volitionary tremor. That this path is specially liable to be affected is well seen in these cases here described, as in most of them patches were found at many different levels. They occur very frequently in the superior cerebellar peduncles, in the red nucleus, and in the optic thalamus.

The relation of the symptoms to the areas of sclerosis found after death is well seen in Case I. (L. W.). The symptoms in this case began with weakness of the legs, which passed off, recurred, grew rapidly worse, and terminated in a spastic paraplegia. This is obviously the clinical manifestation of the dense areas of sclerosis which were found throughout the spinal cord. These were obviously of long duration, and seem to have been the earliest manifestation of the disease. The patches which were found in the sacral region were also of an early date, and were responsible for the affection of the sphincters from which she suffered. The spinal cord lesions in this case were thus very extensive and appeared at an early stage of the disease.

The next symptom of importance to appear was the volition tremor, with which we associate the patches in the superior cerebellar peduncle, red nucleus, and optic thalamus. These appeared also at a fairly early stage. The nystagmus was also an early symptom, and was undoubtedly associated with the peri-ventricular and peri-aqueductal sclerosis, only parts of the oculo-motor nucleus being involved.

After a short time the symptoms followed each other rapidly. An extension of the patches in the cervical region caused sudden numbness in the left arm. This was followed by a rapid development of large patches in the pons, one of which, involving the eighth nerve, produced deafness in the right ear, and another, catching the facial nucleus, produced a right facial paralysis. These patches were found in an early stage and thus appeared late in the course of the disease. An extension still later of a patch involved the sixth nucleus, and led to diplopia from paralysis of the external rectus muscle. This was soon followed by the medullary patches which involved the twelfth nerve and nucleus, and led to a protrusion of the tongue to one side and slight difficulty in speech and swallowing. Later the vision suddenly became dim, and next morning she became totally blind. This was obviously the result of the development of patches in the optic tracts and chiasma. Later the areas in the spinal cord became still more extensive, and the effects of a lower neurone

lesion became evident, resulting in muscular wasting and emaciation.

Description of Plates.

The illustrations are grouped together to follow as closely as possible the different sections of the histological study. Plates I. to VIII. show the general topographical distribution of the areæ in myelin sheath stain (the Kulschitzky-Pal modification of Weigert's medullated sheath stain (I. to VII.) and Marchi method (VIII.)). In the remaining illustrations chief stress has been laid upon the evolution of the sclerotic areas and upon the sequence of the changes in the individual tissue elements.

Plates I. and II.—Areæ in cerebral hemispheres at different levels.
Plates III. and IV.—Areæ in the brain stem and optic nerves, chiasma, and tracts.
Plates V. and VI.—Areæ in the spinal cord at various levels.
Plates VII. and VIII.—Special features of cerebral and spinal cord areæ.
Plates IX. and X.—Evolution of an actual sclerotic area through a stage of tab-granule cell formation; sections cut in longitudinal and transverse direction of nerve fibres.
Figs. 59 to 61.—Glia cell changes in the respective layers of the cortex.
Figs. 62 to 64.—Ganglion cell changes in cortex and anterior horn of grey matter.
Figs. 65, 66.—Negative and positive pictures—myelin sheath and neuroglia stains.
Figs. 67, 68.—Changes related to axis cylinders.
Figs. 69, 70.—Changes related to blood-vessels.

PLATE I.

Figs. 1, 2.—Complete horizontal sections through the cerebral hemispheres near the base of the optic thalami. *a*=periventricular sclerosis around the posterior horns of the lateral ventricles, whence the sclerosis is continued backwards towards the calcarine fissure; *b*=affection of anterior horns; *c*=of the genu of the corpus callosum; *d*=areæ in the path of the optic radiations; *e*=two early areæ in the retro-lenticular portion of the internal capsule; *f*=in the right parietal operculum; *g*=in the left middle temporal convolution.

PLATE II.

Fig. 3.—Horizontal section through the left cerebral hemisphere near the vertex. *a*=area in a convolution of the intraparietal sulcus; *b*=in the upper frontal gyrus.
Fig. 4.—Horizontal section through the right cerebral hemisphere above the roof of the lateral ventricle. *a*=areæ in the central white matter—probably an extension of the periventricular sclerosis of the roof of the lateral ventricle.

PLATE III.

Figs. 5 to 8.—Taken from Weigert serial sections of medulla oblongata and pons. × 2. Note the distribution of the areæ in relation to the floor and walls of the fourth ventricle; the involvement of the dentate nucleus; the frequent sharp detention of the areæ, that several are surrounded by a zone of shadow sclerosis, which has also a sharp outline; and that nearly all the cranial roots enter into or emerge from demyelinated tissue.
Fig. 5.—Upper medulla continuous with the cerebellum. *a*=entering eighth nerve on each side; *b*=demyelination of vermis and accompanying nodules; *c*=involvement of the hilum of the dentate nucleus on both sides.
Fig. 6.—Junction of medulla oblongata and pons. Note the involvement of the fibres of the inferior and middle cerebellar peduncles on both sides, with extension to the centre of the corresponding flocculus. *b*=area in the cerebellar white matter.
Fig. 7.—Junction of pons and mid-brain. *a*=area in the middle line, which reaches almost to the frontal fillet and cuts across in sharply defined lines, the intersecting fibres of the raphe and the aqueduct, fibres on each side. In the immediately adjacent section there was complete retention of the axis cylinders across this demyelinated area.
Fig. 8.—Slightly above (fifty serial sections) Fig. 7. *a*=zone of shadow sclerosis around area.
Fig. 9.—Mid-brain. × 2. Note involvement of both red nuclei, *b*=extension to the substantia nigra and point of exit on each of the third nerves on both sides.

Fig. 1[illegible] [illegible]

PLATE IV

Fig. 11[illegible]
Fig. 12[illegible]
Fig. 13[illegible]
Fig. 14[illegible]
Fig. 15[illegible]

PLATE V.

Fig. 17[illegible]
Fig. 18.—× 6. Transverse section of spinal cord. Fifth lumbar segment. Note *a* = involvement of
Fig. 19[illegible] [illegible]

PLATE VI.

Fig. 20[illegible] [illegible]
Fig. 21[illegible] [illegible]

PLATE VII.

Fig. 32[illegible]
Fig. 33[illegible]
Fig. 34[illegible]
Fig. 35[illegible]
Fig. 36[illegible]
Fig. 37[illegible]
Fig. 38[illegible]
Fig. 39[illegible]
Fig. 40.—× 13. Convolution of the central fissure; well-defined area in the medullary ray and transition zone.

PLATE VIII.

Figs. 41 to 46.—Marchi-stained sections.

Fig. 41.—× 6. Small "early" area in the posterior columns of the spinal cord. Fat-granule cells, stained black with osmic acid, permeate the tissue, and surround the capillary and larger vessels.

Fig. 42.—× 6. Extensive distribution of the areas containing fat-granule cells, involving almost the whole transverse section of the cord.

Fig. 43.—× 5. Longitudinal section of cord showing the interrupted lines of fat-granule cells.

Fig. 44.—× 30. Margin of cord with pia showing the lateral vessels, with fat-granule cells in their adventitial lymphatic sheaths, passing towards the inner layers of the pia, within which they spread in all directions.

Fig. 45[illegible] Small "early" area in the central white matter of the brain; the tissue around the central short vessels is permeated with fat-granule cells.

Fig. 46[illegible] Demyelinated "early" area at the tip of a medullary ray, extending through the radiations [illegible]

PLATE IX.

Figs. 47 to 52.—Evolution of an actual sclerotic area, in the posterior columns of the spinal cord, through a stage of fat-granule cell formation. Sections are cut in the longitudinal direction of the nerve fibres. *a* = glia nuclei; *b* = glia fibrils; *c* = fat-granule cells; *d* = persistent axis-cylinders; *e* = blood-vessels; and *s* = dense sclerotic tissue.

Fig. 47.—Low-power view of "early" area in stage of so-called "fat-granule cell myelitis." Van Gieson's stain. × 70.

Fig. 48.—Fat-granule cell formation, glia cell proliferation, and glia fibril formation at the expense of the glia cell protoplasm and protoplasmic processes. Heidenhain's iron-hæmatoxylin. × 200.

Fig. 49.—Increasing glia fibril formation, with gradual removal of fat-granule cells. Heidenhain's iron-hæmatoxylin. × 200.

Fig. 50.—Complete sclerosis—longitudinally coursing glia fibrils. Palladium methyl-violet. × 200.

Fig. 51.—Complete sclerosis—numerous retained axis cylinders (darker lines) surrounded by parallel-coursing fine glia fibrils. Methyl-violet. × 200.

Fig. 52.—Complete sclerosis—tissue consists of glia fibrils, a few retained axis cylinders, and blood-vessels. Van Gieson's stain. × 70.

PLATE X.

Figs. 53 to 56.—Evolution of an actual sclerotic area through a stage of fat-granule cell formation. Sections cut transversely to the direction of the nerve fibres (*cf.* Figs. 47 to 52). Heidenhain's iron-hæmatoxylin. *a* = glia cells; *b* = glia fibrils; *c* = fat-granule cells; *d* = axis cylinders; *e* = "Kudstreifen."

Fig. 53.—Stage of glia cell proliferation and fat-granule cell formation. × 370.

Fig. 54.—Stage of commencing glia fibril formation. × 300.

Fig. 55.—Stage of advancing glia fibril formation. × 300.

Fig. 56.—Stage of complete sclerosis—a dense tissue with very fine meshes. × 60.

Fig. 57.—Glia cell changes in the posterior columns of cord; rows of large, frequently multi-nucleated protoplasmic glia cells. Heidenhain's iron-hæmatoxylin. × 500.

Fig. 58.—Glia cell changes in the deepest layer of the cortex. *a* = proliferated glia cells with numerous fibrils; *c* = ganglion cells; *e* = blood-vessels. Note the relation of the glia cell processes and fibrils to the capillary walls. Methyl-violet. × 600.

PLATE XI.

Figs. 59 to 61.—Glia cell changes in the respective layers of the cortex. *a* = proliferated glia cells with numerous fibrils; *b* = nests of small glia cells around the ghosts of ganglion cells; *c* = ganglion cells; *d* = persistent axis cylinders; *e* = blood-vessels. Note relation of glia fibrils and glia cell processes to the capillary walls.

Fig. 59.—In the polymorphous (deepest) layer. Methyl-violet. × 200.

Fig. 60.—In the layer of the deep pyramids. Methyl-violet. × 200.

Fig. 61.—In the granule layer. Hæmatoxylin and eosin. × 200.

Figs. 62 to 64.—Changes in areas in the grey matter.

Fig. 62.—Demyelination of cortex without any change in the cyto-architecture. Van Gieson's stain. × 60.

Fig. 63.—Demyelinntion of cortex with marked glia cell reaction in the transition zone and in the deep layers of the cortex. Note the marked alteration in the Betz cells (*a*). Van Gieson's stain. × 60.

Fig. 64.—Ganglion cell changes in the anterior horn of the spinal cord in a de-myelinated area; cells very atrophic, but rounded forms present with nucleus and chromophile granules almost normal. Polychrome methylene blue. × 80.

PLATE XII.

Figs. 65, 66.—Low-power view of areas, in myelin sheath and glia stains respectively, to show comparative negative and positive pictures.

Fig. 65.—Area in central white matter to show absence of myelin. × 40.

Fig. 66.—Similar area to show proliferation of glia in the demyelinated tissue. × 60.

Fig. 67.—Persistence of swollen axis cylinders in cross section of area in posterior columns of cord. Bielschowsky-Williamson silver method. × 200.

Fig. 68.—Persistence of axis cylinders in optic nerve which was completely gelatinous and demyelinated. Cajal's silver method. × 200.

Fig. 69.—Transition blood-vessel in the cerebral white matter—to show the cell content of the adventitia during the stage of advancing sclerosis. Van Gieson's stain. × 370.

Fig. 70.—Condensed "hyaline" vessel in cerebral white matter, the outer layers of the adventitia (*b*) still separated. Note the lessening cell content of the vessel wall. Van Gieson's stain. × 370.

OBITUARY.

SIR LAUDER BRUNTON, BART., M.D., C.M., D.Sc.(Edin.), M.D., R.U.I.(Hon.), LL.D.(Edin. and Aberd.), F.R.C.P., F.R.S., Consulting Physician to St. Bartholomew's Hospital.

UNDER the ever-swaying and resistless scythe of the Great Reaper, the Old Guard—the Medical Graduates in the sixties of last century—are rapidly passing on; the last, and one of the most distinguished of them all, to go is Sir Lauder Brunton. Seventy-two years ago, in the quiet village of Bowden, sheltered by the tripartite Eildons, Thomas Lauder Brunton was born, the youngest son of James Brunton of Hilton's Hill. He came of a good and vigorous Border ancestry, through whom ran a notable thread of genius, and of this endowment Lauder had a part. His early education was in private, but he finished it at the Queen Street Institution, and in Edinburgh in 1861, through a mutual friend, a schoolfellow of Brunton's, we first met. Friendship quickly followed, for we were both sons of the Border—a district in which there exists a kindly and clannish feeling—and the sensitive plate of memory still holds the portrait of the short, well-knit lad, with features cut like a classic cameo, with bright, earnest eyes in which there ever lurked a merry twinkle, with shy and diffident manners, little interested in games, but diligent in all class work, especially—and thus evidencing his early bent—in chemistry and physics. In 1862 he began his medical studies, and he soon made his mark, as much by qualities which naturally gained the esteem of his fellows as by the position he attained in his classes. A brilliant Scottish author has written: "If your associates attach a diminutive to your Christian name, you possess something which millionaires would gladly give half their fortune to purchase." Brunton had this possession, for to many, and to all his intimates, he was "Tommy Brunton." He was greatly distinguished in all his classes and gained many honours, for, in addition to his marked abilities, he had an extraordinary capacity for work. The greatest proof of this lies in the fact that in his last year—and the curriculum was then a four-years' course, with a final examination in the seven most important subjects at its close—he wrote an original thesis upon "Digitalis and Certain of its Effects upon the Urine"—necessitating many experiments made upon himself, as well as those conducted in the laboratory. For four months he weighed all he ate and drank, and made on an average at least three qualitative examinations of his urine daily, in order to determine the effect of the drug upon that secretion. This was only one aspect of the inquiry, for digitalis had long been known to possess a powerful effect upon the circulation, and it was in the way in which this was produced that lay

his chiefest interest. He was a pioneer in the methods by which he carried this out, for in these now distant days the clinical instruments available by the diagnostician were few; for I recall, when Clinical Clerk in the Fever Wards under Warburton Begbie, the visits of Frederick Taylor, who, with a thermometer nearly ten inches long and placed in the axilla, set himself sometimes on his knees by the bedside of the sufferer from typhus in order to ascertain the height to which the column of mercury rose, for there was then no self registering instrument, and it was the first time that in Edinburgh such observations had been made.

Brunton, in the laboratory of Sir Douglas Maclagan, was working at the subject of the blood-pressure of animals, to whom he had given digitalis, with a similar simple column of mercury, although, happily, he possessed a sphygmograph with which to take his own pressure. By these means he was able to determine that digitalis not only increased the muscular power of the heart, but by its contractile action on the arteries raised the blood-pressure. For this thesis, fraught with high promise, he received, upon his Graduation with Honours, in 1866, a Gold Medal.

As marking his distinguished undergraduate career, he was now appointed Resident Physician in the Clinical Wards of the Royal Infirmary, at that time under the charge of Professors Hughes Bennett and Douglas Maclagan. This was for him a happy combination with which to work, for Bennett was a great physiologist as well as a most thoughtful physician, and Maclagan, with a wide knowledge of pharmacology and experimental investigation, was an excellent clinical teacher. It was while under their combined influence and the knowledge that the highly gifted Arthur Gamgee and laboratory research had taught him, that, when in a severe case of angina pectoris, in which, by the use of the sphygmograph, he detected the existence of high blood pressure, he called to his aid the dilator action of nitrite of amyl, thus bringing relief to his patient, and through him to many a similar sufferer, and discovering a potent remedy which was then sadly wanted. This was a fine example, as he himself maintained, of the benefit of experimental work when correlated with clinical data, and through it he established the employment in practice of vaso-dilators. With some of the most formative of his years behind him, with a great capacity of acquiring knowledge, and with the resolve to lay the strongest possible foundation for his future work, he spent nearly two years in several great continental laboratories and cliniques, in at least one of which — that of Leipzig — he gained much distinction. He became M.D. in 1868, and crowned his scientific studies by obtaining a D.Sc. (Physiology) in 1870. In 1870 the magnetic action of London, which draws so many of the young *élite* of all professions within its orbit, fixed Brunton's future, and, settling there, he became

Sir Lauder Brunton, Bart.

a member of the Royal College of Physicians, and a year or so later was elected Casualty Physician and Lecturer on Materia Medica at St. Bartholomew's Hospital. His wish—born of his scientific studies and his knowledge—to be a teacher in a great medical school was now gratified, and he set to work with all the immense energy he possessed to carry out his purpose, which was to infuse a new life into the teaching of Materia Medica and to make pharmacology a science. He established a laboratory in which research work could be carried out, and at much personal effort and expense he maintained its working. The results obtained he gave forth from time to time through various channels, and its summation appeared in 1885 in his *magnum opus*, *Pharmacology, Materia Medica, and Therapeutics*. The merit of this book was quickly recognised, and it passed rapidly through several editions as well as translations into other languages, and established his reputation as a scientist a reputation foreshadowed by the fact that, at the early age of thirty, he had been elected an F.R.S. From 1875 onwards much valuable material came from his facile pen, bearing chiefly on the subjects which always interested him most—*The Action of Medicines*, *The Disorders of Assimilation* and *The Therapeutics of the Circulation*; while the delivery of "The Gulstonian" and "Croonian Lectures" at the Royal College of Physicians gave opportunity for an explanation of his methods and of his views on "the connection between chemical structures and physiological action in the prevention, control, and cure of disease." Of these lesser publications *The Action of Medicines* will probably remain as the happiest example of his literary style, his eminent ability, and the extraordinary breadth of his knowledge, and will be the most helpful to the practitioner. It was fitting that in 1894 he should deliver the Harveian Oration, for his best work had its basis in physiology, of which the great anatomist was the founder, and it was natural that problems connected with the circulation should form its subject. In 1889 he was asked by the *Lancet* to go to Hyderabad in order to repeat some experiments upon the administration of chloroform which had already been carried out by a committee under the Nizam's Government. The originator of this committee was probably the late Colonel Edward Lawrie, I.M.S., then the Residency Surgeon. He was, although somewhat junior, one of our fellow-students a man of brilliant parts, and one who had well-nigh implicit faith in the safety of chloroform administration, but who, while house-surgeon to Mr. Syme, had been somewhat obsessed by that great surgeon's oft-repeated *dictum*, that "he had never had a death from chloroform." In this Fortune had been kind, for it was not the experience of his surgical colleagues or that of all his own house-surgeons. Brunton went to India, and carried through his mission with all skill, and care, and thought, as was his wont, but arrived at no definite result,

except this, that as regarded himself he had strong faith in the safety of chloroform. On several occasions I witnessed its administration to him, and the confidence with which he took it was remarkable. Disappointing in its results, as his report seemed, it added to his growing reputation, for he was now one of the best known physicians in London. Dr. John Brown, the author of *Rab and his Friends*, and of much else that is memorable in Scottish literature, loved to descant upon the primary qualifications of a physician, and he summed them up in four words—capacity, perspicacity, sagacity, and efficacity—this last the will and the way to turn all the others to account when called upon. Brunton possessed in good measure all these, and yet he lacked somewhat of a fifth and great qualification—that intuitive instinct—which was so notable in Jenner, in Gull, and in Gee; but he was so wise, so clear in judgment, so full of knowledge, so rich in experience, and so careful in physical examination that he attained his end, and made many notable diagnoses. Moreover, he had that *nous*—that presence of mind—which made him singularly resourceful and helpful in suggesting lines of treatment in emergency and rendered his presence often a benediction. His common-sense philosophy and his power of adapting his scientific knowledge to the affairs of common life, coupled with his intense sympathy and consideration, gained for him the perfect confidence and the warm affection of his patients, and they forthwith became his friends, and in friends he ever found the sunshine of his life. Honours, as he well deserved, came to him freely, for in 1900 he became a Knight; in 1904, after twenty-nine years of devoted and unstinted service, he was elected Consulting Physician to his hospital; and in 1909 he was made a Baronet.

As it is always the busiest men who shoulder the extra burdens, he did not confine his abilities and his energies to the exercise of his profession, for he displayed a warm and helpful interest in matters of national importance—Physical Education and Hygiene—and in his latter days he did much, and most unostentatiously, to help the soldier in the field. Great as his success was, he did not escape the thunderstorms of life, and twice or thrice in his very heyday he was struck down by severe illness, and laid aside for considerable periods. The most severe of these occurred some fourteen years ago, when he contracted a grave attack of septicæmia—the result, he believed, of inoculation in a hospital ward through a soiled towel. After a time of much suffering and danger in England, he passed to my surgical care in Cannes, and impressed me greatly by his extraordinary attitude of patience and fortitude under constant pain during the many months he was there. Long time as I had known him, it was in these circumstances, and in anxious days, that the man stood fully revealed, and the revelation was that of one—and I use the ultimate words applied to human character—noble, generous, and courageous. He

was noble in the sense that self sacrifice and duty were the watch words of his life, and that in all he thought and did he was unselfish; he was generous in his kindly attitude to others—in his warm gratitude for all that was done for him, and in the help, through time and money, that he gave to many; and he was courageous—and this through Faith—in regard to his sufferings and to all that lies Beyond. He lived a very full and laborious life, in which one of the precepts governing his daily round, and which he often repeated, was "work, for the night cometh when no man can work"; he did much good in many ways in his profession and in the world outside of it, and he leaves an enduring memory as a "beloved physician," and the first of scientific pharmacologists. His home life was a very happy one, for he lived most deeply through his affections, and Lady Brunton, whom he married in 1879, brightened, and sweetened, and made him more lovable; while her death, after thirty years of most helpful companionship, did him grave physical harm, for it was just after this event that the first evidence was detectable of the lesion which was to end his life. "A broken heart" is no mere figure of speech. The death, a year ago, of his youngest son—a man of much promise—a surgeon in the Guards—in action, was a cause of further sorrow; but this he regarded in the light of a short separation, and he judged rightly. He is succeeded by his eldest son, Lieutenant J. S. L. Brunton, at one time an officer in the Royal Artillery, now in the Canadian Army, and there proving by his loyal actions his sonship to a patriot father. In a fine appreciation in the pages of the *Lancet*, voicing his profession, these words occur—"The congenial, kindly manner which endeared him greatly to his patients was a true indication of his almost emotional feeling for the suffering of others. No one more bounteous with his time, thought, or money—whether to individual friends or for the public good—ever adorned the medical profession," while I—designated in a line of farewell as "his oldest friend"—after scanning the wide fields of memory of more than fifty years, would lay reverently and affectionately this tribute on his tomb:—

"He was perfectly pure, perfectly gentle, perfectly upright, perfectly true, perfectly lovable, and richly endowed with the Spirit of the Master whose he was and whom he served."

J. A. MacDougall

ALEXANDER BALLANTYNE, M.D., F.R.C.P.

The death of Dr. Alexander Ballantyne, which took place at Oxford-holme, Gullane, on 3rd October, removes from our midst one of the most highly respected and generally beloved members of the medical profession in the Lothians. Shortly after his birth, at Chorley in South Lancashire in 1858, his parents moved to Dalkeith, and there he spent

his long and eminently useful life, playing the part, for which he was so well fitted, of doctor, friend, and counsellor, alike to gentle and simple. His influence was not confined to the district in which he practised, for his colleagues in Edinburgh recognised his professional work by electing him President of the Obstetrical Society, and his social camaraderie by asking him to fill the chair of the Harveian Society. On the golf links, where he spent such time as he could spare from a busy practice, his skill, his sportsmanship, and his characteristic old-world courtesy made him a general favourite.

After graduating M.D. in the University of Edinburgh in 1860, he acted for a time as resident physician at Morningside Asylum, and thereafter became assistant to Dr. Anderson of Selkirk—a famous man in his day. With a number of Edinburgh men, including Thomas Lauder Brunton, whose passing we record along with his own, he went to Vienna and spent over a year in post-graduate work. On his return he settled in practice in Dalkeith, and for over forty years his life was spent in the service of the community in and around that town, as family doctor and as medical officer of health.

His energies were not confined to purely professional matters, and he did much to brighten the lives of the people in other directions. His love for music led him, with the aid of his wife, to found the Dalkeith Philharmonic Society in 1875, and he also took an active part in the local Scientific Association. Largely through his efforts the Dalkeith-Newbattle Golf Club was organised, and in 1896 he was elected the first captain of the club. In his day he was also a keen volunteer, acting as Surgeon Lieutenant-Colonel to the old Dalkeith Rifle Volunteers (6th Battalion of the Royal Scots).

Dr. Ballantyne's last days were saddened by the loss of his third son, Commander Ernest Ballantyne, who went down with his ship, H.M.S. *Vidmar*, soon after the outbreak of war. He is survived by his widow and two other sons, one of whom, Dr. Harold S. Ballantyne, has succeeded him in practice in Dalkeith.

JOHN ROGERSON HAMILTON, M.D.

An Appreciation.

"The leaves of Life keep falling one by one."

So sang old Omar, the Persian poet, well-nigh a thousand years ago, and the present autumn has borne witness, in our own profession, to the eternal verity. Recently we had to mourn the loss of Dr. Ballantyne of Dalkeith, and now an equally well known and esteemed Lowland practitioner—Dr. Hamilton of Hawick—has passed to his

well earned rest. He died, as he would have wished, in harness, and was mercifully spared a long illness, which would have been almost unendurable to one of his active and ardent nature. He had been resident in the town of his adoption nearly forty years, and had become, as it were, a part of it; and his kindly presence and genial smile are things its inhabitants will greatly miss. He was a man of very active habits. An early riser, he would often journey thirty miles by train, ride a good distance over the Border uplands, and be back in time to do a long forenoon's visiting in town. He was an enthusiast in his profession, and possessed one of the most optimistic temperaments it were possible to imagine. And while this might, perhaps, on occasion lead him to conclusions scarcely justified by the facts, it was, from his patients' standpoint, an admirable asset, and one which helped many a lame dog over the stile. He was no great believer in drugs, and his prescriptions were of the simplest. Ever loyal to the memory and the methods of the great teachers of the Edinburgh School, he would often sustain his own opinions by what was to him a final *dictum*: "That's what Joe Bell would have done." He used to pride himself upon the regularity and punctuality with which he kept his professional engagements—and justly so. In an experience extending uninterruptedly over twenty years the writer generally found himself second to arrive, and only recalls one solitary occasion when his colleague failed to appear—a truly extraordinary record when one remembers the many possibilities in a doctor's day. He was a man of very forceful personality, and though at times he was intuitive more than logical in argument he had a wonderful way of achieving his purpose. His influence among his fellow-practitioners, both in the town and the wider Border district, was great, and all directed towards good-fellowship and unity.

Outside his personal professional duties he had one great consuming interest—medical politics. No amount of time or energy was too great in compassing work for the British Medical Association. He would think nothing of journeying up to London after a hard day's work, attending a Council or Committee meeting, and returning the following night in time to do another on arriving home. Indeed, it would be an interesting thing to know, were it possible, how many thousand miles he travelled, say during the past five years, on Association business. He was wont to tell, with pardonable pride, of the number of years during which he had had no holiday beyond the annual meeting of the Association. That he rarely missed, and it seemed invariably to invigorate him. But after this course of unwisdom had lasted nearly thirty years he, too, found the necessity for rest as well as change.

A man of a most lovable disposition, his kindness of heart was beyond expression. He was beloved by his adult patients. A certain simplicity in his nature greatly endeared him to children, and perhaps

the happiest part of his last happy day amongst us was that spent in watching a children's party at his own house.

While the exigencies of his busy life left him little leisure for reading, he was a great lover of Scott, and works like Johnson's *Rasselas* and Goldsmith's *Traveller* — better known to a former generation than to this—were sources of unfailing intellectual pleasure to him.

There is among the poems of the late Sir Noël Paton a lovely sonnet, "Timor Mortis Conturbat Me." In it he gives expression to his disappointment at the non-accomplishment of his artistic ideals :—

> "to yield my breath
> Life's purpose unfulfilled! -This is thy sting, O Death."

Little did he dream that this poem was the

> "one Song that should survive
> The singer's voice,"

and that, by winning its way into the anthologies of his native land, it would be a very wreath of amaranth for its author.

The subject of this memoir had neither the ideals nor accomplishments of Art. His life's purpose involved no creative effort. It was to employ his gifts in the interests of his fellow-men ; and none can say it was not fully accomplished. He now rests from his labours and his works do follow him. C. J. W. D.

RECENT ADVANCES IN MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

W. T. RITCHIE, M.D., EDWIN MATTHEW, M.D., J. D. COMRIE, M.D., AND A. GOODALL, M.D.

Pyorrhœa Alveolaris.

Pyorrhœa alveolaris is reviewed by Roddy, Funk, and Kramer (*New York Med. Journ.*, 2nd September 1916), although, as their communication summarised 1496 cases, it is obvious that they must include under this head comparatively mild cases of alveolar suppuration, more commonly known by such names as gingivitis, alveolar periostitis, etc. Taking the term pyorrhœa alveolaris in this sense, they find that it is not a specific disease, that it is caused by an excessive bacterial flora of the mouth, together with abnormalities of the affected tissues due to disease. Oral sepsis, they consider, always precedes pyorrhœa, and the more serious condition may be prevented by regular cleansing of the

mouth and teeth. They assert that there is no specific method of treatment. In reference to the recently advocated treatment of the condition by emetine, they found an amœba present in seventeen cases, and in each of these they administered the drug freely by subcutaneous injection, by injection into the peridental pockets, as a mouth-wash, and also internally. All the cases, which had previously showed amœbæ, failed to show these after one week of the emetine treatment. The amœbæ, however, usually returned in from two to six weeks after the emetine had been discontinued, and the various bacteria in the mouth were not in the least affected, nor did the symptoms diminish under the emetine. Emetine and autogenous vaccines the writers recommend, therefore, on the principle that they may help and cannot do any harm; but the procedures on which they place reliance are regular cleansing of the mouth and teeth, whatever dental treatment may be indicated, and medical treatment of whatever general symptoms or diseases may co-exist.

Pharmacology of Emetine.

The pharmacology of emetine is studied by Pellini and Wallace (*Amer. Journ. of the Med. Sci.*, September 1916), on account of the considerable amount of interest that has attached to this alkaloid owing to its striking results in the treatment of amœbic dysentery and its widespread use in pyorrhœa alveolaris. Its employment in the latter disease was due to Bass and Johns, who have assumed that the entamœba buccalis is an important causative factor in the disease and that it is affected by emetine in the same specific way as the amœba histolytica. Vedder has shown conclusively that emetine is strongly amœbacidal to all forms of non-pathogenic amœba in the dilution of 1 in 100,000. Rogers demonstrated that an equal toxicity is shown against pathogenic forms. Weaker solutions were found to exercise a definitely deleterious action on the parasite. To obtain a 1 to 100,000 solution in the blood it would be necessary to have about 0·05 grm. of emetine in the circulation at one time. Though this is in excess of the dose given for therapeutic purposes, Pellini and Wallace found that emetine is excreted in greatest amount by the stomach and intestines, and therefore exerts its chief effect on the alimentary canal. They found, however, certain deleterious effects. Thus it depresses and may eventually paralyse the heart; it is a powerful gastro-intestinal irritant whether given by the mouth or in subcutaneous injection, and finally it causes a definite derangement of metabolism, characterised by an increase in nitrogen loss and an acidosis.

Gastric Function in Tuberculosis.

Mohler and Funk (*ibid.*) consider that the importance of a study of the gastric function in pulmonary tuberculosis can scarcely be over-

estimated when we consider the great frequency with which symptoms of disordered function occur: thus they found that out of 1000 consecutive patients with pulmonary tuberculosis in various stages symptoms of gastric derangement were present in 64·6 per cent. They found that both motility and secretion were affected from the very beginning of the disease, and they consider that the so-called pre-tubercular dyspepsias are really manifestations following on the tubercular infection. They believe that an irritative stage causing hyperacidity is rare, the type of disease being usually a depression of the stomach functions from the very beginning, with the formation of the clinical syndrome known as "delayed digestion." The visceroptosis and gastrectasis play an important part in the cause of the symptoms, but the swallowing of tuberculous sputum is also a very definite causal factor.

Effect of Diet on the Skin.

Strickler contributes (*New York Med. Journ.*, 9th September 1916) a review upon the relation of diet to some diseases of the skin. Psoriasis has been shown to bear certain relations to metabolism; thus on a given protein dietary a psoriatic subject eliminates less nitrogen in the urine than a normal subject on a similar diet; also such patients show marked nitrogen retention. A low nitrogen diet has therefore a most favourable influence on the eruption of psoriasis, particularly if the latter is extensive, and at the same time it probably renders local remedies more effective by making the skin less irritable. Eczema is due to various causes, but some cases are of dietetic origin, and the diagnosis of these cases can be effected by the anaphylactic food-test. Numerous persons have carried out such investigations, usually by means of the endermic method. A dental burr or similar instrument is used to produce the skin abrasion, and then various suspected foodstuffs are rubbed into the raw area, which, in positive cases, should show a papule at the end of half an hour, followed by erythema. In a large percentage of cases a correction of diet is indicated, and, when effected, is productive of good results. In acute urticaria this anaphylactic food-test is of value from the view-point of both prophylaxis and therapeutics, while in chronic urticaria its value is questionable. In acne vulgaris, on the contrary, food sensitisation does not play a *rôle* in the causation, and in most cases there is an intestinal toxæmia.

Skin-Test in Vasomotor Disturbances of the Upper Air Passages.

Goodall, who has made a considerable study of the skin reaction in the last two years, contributes an article on its value in the management of asthma and allied conditions (*Boston Med. and Surg. Journ.*, 10th August 1916). An individual sensitised to a given proteid may

exhibit a characteristic reaction if the proteid is brought in a soluble form in sufficient concentration into contact with a scratch on the skin. The proteid to be tested is applied to a scratch on the skin of the arm. After 5 to 15 minutes the positive reactions are indicated by varying degrees of local disturbance, which may be ranged in order of intensity as follows: In some cases the first perceptible alteration consists in a sharply circumscribed white area, not elevated, bordering the scratch for a distance of one to three millimetres. In other individuals the first manifestation is a slightly reddened raised area. In more pronounced disturbances the area of swelling is more extensive, consisting even of an œdematous area of two or three centimetres in diameter with a surrounding zone of a centimetre or more of hyperæmia. In the course of 30 minutes, or at most an hour, the reaction fades. The occurrence of this skin reaction may generally be regarded as indicative of sensitisation to the proteid employed. The material is prepared in the case of animals by making extracts from skin, hairs, feathers, etc., with 12 per cent. alcohol, filtering and using the particles suspended in the filtrate; in the case of animal foods juice is expressed and desiccated by a blower; other foods, like egg albumin, casein, cocoa, etc., are obtainable in a desiccated form; pollen from plants is procured by washing the flowers in carbon tetrachloride, straining through muslin, and drying. The reaction is of value in determining the nature of the animal or vegetable substance, food, etc., to which attacks of asthma, coryza, etc., in susceptible individuals are due. As regards treatment, when the cause is found to be seasonal, immunising treatment by the injection of pollen extracts is likely to prove of service. If the cause is perennial and is due to inhalation of foreign particles, as from domestic animals, it is wiser to avoid the cause than to seek a cure by immunisation, because attempts to secure tolerance by injection of horse serum, for example, in cases of horse-asthma have mostly failed. If the disturbing proteid enters into food-stuffs, on the contrary, a tolerance may be gradually established by feeding the substance in progressively increasing amounts. J. D. C.

SURGERY.

UNDER THE CHARGE OF

J. W. STRUTHERS, F.R.C.S., AND D. P. D. WILKIE, F.R.C.S.

The Technique of Laminectomy for Spinal Disease and Injury.

Elsberg (*Journ. Amer. Med. Assoc.*, 15th July 1916) describes his experience of the operation of laminectomy, which he has performed in one hundred and fifty cases.

While many methods have been tried for exposure of the cord and membranes, Elsberg prefers, on the whole, a longitudinal skin incision. After separation of the muscles and removal of the spines and laminæ at their bases by rongeur forceps, the laminæ are bitten away with smaller rongeurs of varying size and shape.

For exposure of the spinal canal a defect of at least one centimetre in width should be made, so that all sides of the cord can be examined with the minimum of handling. If the posterior roots are to be divided, the opening may be somewhat smaller: for decompression purposes the opening should be 1·5 centimetre, or preferably 2 centimetres, in width.

If marked exaggeration of the symptoms of spinal disease follow a laminectomy, the cause is usually rough handling of the parts or hæmorrhage into the arachnoid sac. While all parts of the cord are sensitive, the conus and the origin of the cauda equina are specially so, and after operations in the latter region, disturbances of the bladder, rectum, and of the lower extremities are specially apt to occur.

The benefits from spinal decompression in the case of tumours within the canal are fully as great as in the case of cerebral tumours treated by decompression.

When a decompressive laminectomy is done for old or recent fracture, it is important to remove a sufficient number of spines and laminæ. In addition to removing the arches of affected vertebræ it is necessary to take away one or two arches above and below, so that the dural sac may bend backwards in a gradual curve. It is often advantageous to divide a few slips of the dentate ligament at the affected level, in order to give the cord a greater freedom of movement. Division of a few slips of the dentate ligament near their attachment to the dura does absolutely no harm.

When the anterior surface of the cord requires to be exposed, the manipulation is rendered easier if one or two slips of the dentate ligament are divided on either side.

In some cases of spondylitis the intervertebral foramina are diminished in size and the spinal nerve roots are compressed, so that it may be necessary to enlarge the foramina with small rongeurs. In one case of this kind Elsberg was able to free several nerve roots, and thus relieved the patient of severe root pain. When the dura mater is diseased, a portion of the membrane may require to be excised: this excision may have to extend on both sides down to and into the dural openings, especially if root pains have been severe. If the whole thickness of the dura has to be removed and the dura left open the cord should be covered with Cargile membrane and the muscles and fasciæ stitched with extreme care.

Elsberg lays stress on the importance of studying the appearances of the cord in the different pathological lesions met with. Normally

the spinal cord and nerve roots are of a creamy white colour, and the vessels on the posterior surface of the cord are fairly prominent and tortuous. A dead white colour indicates medullary atrophy; a similar colour can sometimes be noted at or below the level of an old injury of the cord. Below an old transverse lesion the cord is usually of a yellower tint than normal. The white cords are often flat, shrunken, and irregular in consistency.

The spinal cord may appear congested in myelitis, in meningitis, in disease of the dura or arachnoid, after a trauma, or below the level of an extramedullary compression.

It is important to differentiate between the congestion due to inflammation and the condition of the cord in which the veins are distended due to compression at a higher level.

In inflammation the cord has a pink colour, due to congestion of all the vessels in the pia arachnoid, and the nerve roots are also congested and often bluish-red. When the congestion is due to pressure above, the veins stand out prominently as dark tortuous vessels on the cord, while the nerve roots are normal in colour.

A marked congestion of all the roots of the cauda equina is evidence of a low grade inflammatory process, and occurs typically in syphilitic disease of the membranes, and in a condition described by Kennedy, Elsberg, and Oppenheim as neuritis of the cauda equina.

In the case of intraspinal tumours the amount of compression of the cord will depend on the size, consistence, and duration of the growth. Large intraspinal growths are usually much softer than small ones. The large growths cause a more diffuse pressure of the cord. The pressure due to the hard small growths is more concentrated, and this explains why small tumours are more apt to cause a complete immediate transverse lesion of the cord. The amount of injury is greater when the tumour lies under a nerve root than when it is placed on the posterior columns.

For exposure of an intraspinal tumour the arches of at least three vertebræ should be removed. If the tumour is attached to the cord, especially in sub pial growths, it is often advisable to leave a piece of capsule attached to the cord. Most spinal growths are encapsuled, grow slowly, and have little tendency to recur. In a series of twenty-eight cases Elsberg found only one extramedullary growth on the anterior surface of the cord. In a series of one hundred and fifty cases of spinal operations the mortality was 10 per cent. In most of the fatal cases the patients' condition was serious at the time of operation.

The Treatment of Pruritus Ani by Subcutaneous Injection of Alcohol.

Whilst pruritus ani is frequently due to obvious lesions, such as hæmorrhoids, fissure, or eczema, or to a constitutional condition such

as diabetes, in other cases the causal factor is obscure, and a successful method of treatment is often difficult to discover.

Various theories — acid secretions, latent organismal infections, central and peripheral nerve disturbances—have been suggested to account for the condition. Numerous methods of treatment have been employed for its alleviation. Ointments, powders, lotions, have been tried with varying, but never constant, success. Cauterisation, X-ray exposures, and vaccines have furnished more recent attempts to solve the problem. Operative measures, such as the Ball and Lynch procedures for division of the peripheral cutaneous nerves, have been employed with success. The success of alcohol injections in producing localised lasting anæsthesia in cases of neuralgia, suggested the application of the same principle for the abolition of unpleasant sensations in the anal and perianal regions. As alcohol produces its effect by causing degeneration of nerve fibres, the subsequent result is much the same as that achieved in Ball's operation, when the cutaneous nerves are divided.

Stone (*Johns Hopkins Hosp. Bull.*, August 1916) records the results of his experimental and clinical experience with this method of treatment.

Since there is no selective action of alcohol one might expect loss of sphincteric control if the injection reached the motor nerves to the anal muscles. Further, the injection, if too superficially placed, might cause a slough and ulceration. The author experimentally tested these theories by injecting alcohol into the anal region of dogs, the depth of the injections being varied, and determined the following facts:—Alcohol injections will produce complete local anæsthesia. If injected deeply enough sphincter paralysis and incontinence are produced. If introduced into the skin itself, superficial sloughs are caused. It is quite possible, however, to produce anæsthesia without either of these undesirable complications. The method has been tried clinically during the last two years in seventeen patients. The facts observed are as follows:—There is not much pain associated with the injection. There is some soreness during the first twenty-four hours, and thereafter the only subjective sensation is numbness. There were several cases in which the technique was faulty, and small superficial sloughs developed, which, however, all healed promptly and without any difficulty. The author has not yet established the likely duration of the anæsthesia, but one patient required a second injection eight months after the first. All of the patients were much gratified at the relief from itching. The appearance of the skin shows great improvement after the treatment. The thickened folds and ridges become less pronounced, the colour becomes more normal, and the scratch marks disappear.

The technique of injection is simple: the area affected is carefully noted from the patient's description. Under general or local anæsthesia the injection is then made so that the whole area is anæsthetised. One per cent. novocain solution, or a similar strength of quinine and

urea hydrochloride, has proved quite satisfactory. The syringe is filled with alcohol (95 per cent.), and the usual fine hypodermic needle is employed. The needle is inserted vertically through the entire thickness of the skin, and then is inclined sharply to the side, so that it lies nearly parallel to the skin surface. When the needle is properly inserted in the subcutaneous fat it can be moved fairly freely from side to side under the skin, and can be felt moving with the finger placed over it. If this freedom of movement is lacking, the needle is probably engaged in the corium, and, if an injection is thus made, the skin will probably slough. With the needle properly placed, the whole area involved is injected, and enough alcohol should be introduced to underlay the skin thoroughly. The injection may be carried up to the margin of the anus, but the author has never found it necessary to inject the anal canal itself.

As compared with the operative treatment of pruritus, the method of alcohol injection has certain advantages. It is safer, quicker, requires no after-dressing, and entails no hospital expense. Further, the method is as likely to succeed as is an operation, and there is no undermined skin with impaired circulation and a potential dead space difficult to keep clean.

Choice of Operation in Cure of Aneurysms of the Extremity.

Before deciding on the type of operation which should be performed in the case of an aneurysm of an extremity, it is necessary to obtain as accurate a knowledge as possible of the state of the collateral circulation. Most of the tests recommended are unsatisfactory. Bernheim (*Interstate Med. Journ.*, May 1916) places most reliance on a modification of the test devised by Moszkowicz. This consists in applying first a broad Esmarch elastic bandage from the tips of the toes until the upper pole of the aneurysm is reached. At this level the bandage is held in position by a clamp and fixed, while the parent vessel is compressed in Hunter's canal by means of a pad until all pulsation in the sac is arrested. After five minutes in old subjects, and eight in younger ones, the elastic bandage is quickly removed while the compressor still controls the main artery. Close attention must be paid to the hyperæmic wave, and the progress of the blush is noted as it descends, rapidly at first, in the zone immediately below the level of compression.

If a living colour returns throughout the leg within a few minutes one may conclude that a satisfactory collateral circulation is present, and that a type of operation involving occlusion of the main trunk may safely be employed. When, on the other hand, instead of a blush the limb retains the cadaveric appearance resulting from the application of the bandage, either *in toto* or in part, an operation must be chosen which will leave the main trunk intact or will re-establish it.

The mere recognition of an aneurysm, therefore, is not sufficient to establish a plan of operation. In addition to the test described there are certain other points which, if observed and properly interpreted, will assist in determining the proper method of treatment. In the case of a popliteal aneurysm the normal pulses at the ankle may or may not be present. Their absence usually means that the compensatory circulation is sufficiently developed to sustain the life of the limb. The presence of the pulses at the ankle should put one on his guard, since notice is thus served that there has been no demand for a collateral circulation.

Bernheim describes an operation which was successfully accomplished by transplantation of a segment of a vein to maintain the parent trunk in a case of popliteal aneurysm. In this particular case the tests had demonstrated an insufficient collateral circulation, and the condition of the aneurysm excluded the possibility of performing a restorative or reconstructive operation according to Matas' method. After resection of the aneurysm a portion, 12 centimetres in length, of the internal saphenous vein was inserted in the gap, the ends of the vein being stitched to the divided artery above and below by Carrel's end-to-end suture. In transplanting a segment of vein in order to maintain the continuity of an arterial trunk it is important to reverse the vein in order to obviate the resistance of the valves which would otherwise have to be forced by the arterial pressure.

Bernheim's case is specially instructive, because the operation proved successful in spite of the fact that the artery was thickened at the points of suture. The patient was completely relieved of all his symptoms, and pulsation returned at once, both in the dorsalis pedis and posterior tibial arteries. The patient reported himself some time later after leaving hospital, and then, after a considerable lapse of time, pulsation could be felt all along the vein graft as well as in the arteries of the foot. The result obtained in this case could only have been achieved by the most careful attention to the details of technique in blood-vessel suture. J. M. G.

LARYNGOLOGY, OTOLOGY, AND RHINOLOGY.

UNDER THE CHARGE OF

A. LOGAN TURNER, F.R.C.S., J. S. FRASER, F.R.C.S., AND W. G. PORTER, F.R.C.S.

Abscess of the Lung Following the Operation of Removal of the Faucial Tonsils.

The frequency of the operation for removal of the tonsils by specialists and by general practitioners has probably led the medical profession and the general public to regard the operation as a very minor surgical

procedure. As a natural result of this attitude of mind there is a tendency to look upon the patient who has been operated upon as free from any serious post-operative complication. Though we are unwilling to suggest that an element of carelessness in the after-treatment of these patients has in consequence gradually developed, we are of the opinion that sufficient prudence in dealing with them is not always exercised; further, we think that with the substitution of the complete removal of the tonsil for the older method of tonsillectomy the operation should be relegated to the class of major operations upon the throat, and as such treated with greater respect. This opinion has recently been supported by the publication of a number of cases in which, after the removal of the tonsils, abscess of the lung has developed, in some instances with a fatal result. We are indebted to the profession in America for drawing attention to the subject.

Morris Manges has contributed an important paper, entitled "The Occurrence of Abscess of the Lung after Tonsillectomy, with the Report of Nine Cases in Adults," in the *Journal of the American Medical Association* (May 1915). He discusses the etiology of the complication under the following heads:—(1) Anæsthesia. (2) Aspiration of (*a*) infected blood, (*b*) pieces of tonsillar tissue. (3) Embolism or infarction. (4) Some special infective agent. (5) Some antecedent course, local or general. In a discussion upon the subject these points were dealt with by Coakley in the following way:—The anæsthesia *per se* could not be regarded as the etiological factor. As the cases which have occurred were operated upon under a general anæsthetic, when the danger of aspiration undoubtedly arises, the anæsthesia can only be regarded as having a contributing influence. The aspiration of infected blood must be regarded as the essential cause. Unless the operation is performed in an unskilful manner, there should be no aspiration of portions of tonsils. Most operators must be acquainted with the fact that during the operation cheesy infective material may be squeezed from the tonsillar crypts, and that this may pass into the pharynx and may be aspirated, mixed with blood or saliva, and thus prove a source of pulmonary infection. It is difficult to attribute a single localised suppurative area to embolism or infarction. If the resulting pulmonary lesion were multiple, more weight might be attached to the embolic theory. If the cases that have been recorded had occurred in the hands of one operator, and if the operation had been performed in one particular room or hospital, the question of a local infective agent might be seriously considered, but such has not been the case. An antecedent cause—some condition in the patient himself prior to operation—might be an important factor, were it always possible to determine the existence of such. The examination of the patient before removal of the tonsils is not, as a rule, a very

exhaustive one, and a small tubercular pulmonary focus may readily enough escape detection.

Coakley concludes by calling attention to the care with which such operations should be carried out in order that a suppurative pulmonary complication may be avoided. The operation performed under a general anæsthetic should be undertaken by a man who knows his business, and his assistants should likewise have had proper training. The throat should be carefully sponged to prevent the escape of septic material into the pharynx and to keep it free from blood during the operation. All bleeding should be stopped from the first tonsil before the second is touched, and the patients should not be allowed to leave the house or travel until the wounds are healed.

Richardson (*The Laryngoscope*, St. Louis, July 1916) deals with the same subject. He favours the view that the pulmonary suppuration after tonsillectomy is in all probability due to embolism. At the time of the operation a large number of veins are opened, and he says that these may remain patulous for several days. Septic clots or other septic material may thus be carried into the lungs. The character of the onset leads him to conclude that from a clinical point of view the septic infarct more readily explains the etiology. He admits that tonsillar blood containing cheesy concretion may play a causal *rôle* in a certain number of cases.

He reports three cases of his own in which a pulmonary complication arose, and he refers to two others with which he had been intimately associated. One of his own cases may be recorded here as an example of the condition. A vigorous man, æt. 45, had both tonsils removed. The convalescence was slow. A moderate fever continued for six days. On the seventh day the patient returned to his own house and remained under the constant care of his own physician for nearly ten days. Later, Richardson again saw him in consultation and found him very ill. He had a septic temperature, coughing constantly, complaining of great pain at the base of his right lung, and he presented a very anxious and distressed countenance. A slight area of dulness, diminished breath sound, with some moist râles, could be made out over the base of the right lung posteriorly. Several weeks later he was operated upon at the Mayo clinic, and an abscess was drained. Complete recovery followed. Richardson concludes his paper by drawing the attention of the profession to the possible immediate and remote risks or sequelæ that may follow removal of the tonsils and endanger the welfare, and even the life, of the patient. He does not consider that these sequelæ and complications are always due to the want of skill and technic of the surgeon, and he believes that more frequently they are beyond the control of the operator and wholly independent of the care and attention paid to the post-operative management of the case. He urges a more careful

weighing of all the points which seem to make the operation necessary, eliminating all the other possible sources of infection before the operation is decided upon. While at times the results obtained after removal of the tonsils are brilliant, we may find that they do not always compensate for the risk through which the patient passes. Scudder (*Boston Med. and Surg. Journ.*, October 1915) states that attention has been drawn to lung abscess following tonsillectomy, and in his experience there have been several cases.

In the *Interstate Medical Journal* H. Wessler has published a skiagraphic study of the pulmonary inflammation due to aspiration which sometimes follows tonsillectomy, and the reviewer is indebted to the *Journal of Laryngology* (September 1916) for the abstract which he now quotes. Wessler considers that the term "lung suppuration" is more accurate than abscess or gangrene of the lung, which describe only a possible phase of the process. At the Mount Sinai Hospital, New York, he has examined with the X-rays eight cases of pulmonary suppuration following removal of the tonsils. These formed as high a percentage as 28 of all the cases of pulmonary suppuration observed in the same period. In all of them a general anæsthetic had been administered—a point which is of considerable significance in connection with the etiology of the complication.

After incubation of a few days symptoms of broncho-pneumonia arise, and later there is evidence of suppuration. In practically all the cases the sputum is foul at some time or other. Periods of gangrenous sputum alternate irregularly with others in which there is no distinctive odour. Hæmoptysis is a very constant symptom. Pain is frequent and due to associated pleurisy. A predilection for the right lung was noted in six out of the eight cases, and any lobe may be affected. As a rule an area of dulness of varying extent is demonstrable, with but few changes in the respiratory sounds. In none of the cases were there clinical signs of a cavity, the dull areas being evidently consolidated and poorly aerated lung, sometimes covered by thickened pleura. In six of the cases spontaneous recovery took place in from six weeks to five months. In one case recovery took place after two years, while the remaining case is still unimproved.

Skiagrams show an infiltration of the lung of varying extent. In five cases a cavity was demonstrated, in some with a fluid level which altered with the change of position of the patient. The shape of the infiltrated area varied. In some a lobar distribution was seen; others gave the impression of a residual infiltration involving only the small portion of a lobe. Unless the lower lobe is involved there is no restriction in the movement of the diaphragm. When present, cavities are easily recognised as lighter areas of circular or elliptical shape within the shadow of infiltrated lung. In two cases multiple cavities were found, and in two located at the hilus. When filled with secretion

they may be invisible, but are brought into view after copious expectoration. It is possible to follow the process of cure by means of the X-rays. The infiltration gradually becomes less dense and fades at the periphery until it disappears, leaving perhaps a few thickened strands for some time. Clinical cure, however, may be associated with persistence of the infiltration.

While the reviewer is unaware of the occurrence of any such complication in his own experience, he cannot presume on that account that it has never occurred. In the majority of such operations the patient passes out of his hands after a few days, and the subsequent history of the case is unknown to him. As an illustration of this point the reviewer has become acquainted during the last month with a case of pulmonary suppuration following tonsillectomy. The specialist who performed the operation is still ignorant of the complication which has developed in his patient, who is slowly recovering. The tonsils were dissected out under general anæsthesia, the operation being somewhat prolonged. Pleurisy developed shortly afterwards, necessitating his remaining in the nursing home for a longer period than usual. After the patient had been removed to his own house his cough and expectoration caused some anxiety, and the possibility of tuberculosis was considered. Careful examination of the sputum, however, revealed no evidence of tubercle bacilli, but the micrococcus catarrhalis and the pneumococcus were in considerable numbers. The X-rays demonstrated an area of lung opacity in the region of the left scapula.

An interesting point naturally arises in connection with this class of case, and it is right to make reference to it. The possibility of the aspiration of a tooth detached by the gag or as the result of manipulations in the mouth must not be lost sight of. Such accidents have occurred, and their medico-legal aspect may be the cause of great anxiety to the surgeon. As the subject of pulmonary suppuration has been ventilated in the papers briefly sketched here, it would be fitting to have the subject more thoroughly investigated in the numerous clinics devoted to diseases of the nose and throat.

A. L. T.

INFECTIOUS DISEASES.

UNDER THE CHARGE OF

CLAUDE B. KER, M.D.

Serum Treatment of Poliomyelitis.

Flexner (*Journ. Amer. Med. Assoc.*, 19th August 1916) gives a brief description of the efforts made to secure a satisfactory serum treatment for this disease. Several investigators have noticed that monkeys, which have survived an attack of experimental poliomyelitis, are no longer subject to successful reinoculation with the virus of the disease.

In the blood of such monkeys, and also of human beings who have had the disease, immunity substances have been detected which have the power, if mixed with the virus *in vitro*, of neutralising it. The serum, moreover, of monkeys actively immunised against the disease with the virus, contains similar immunity bodies.

Flexner and Lewis next demonstrated that immune human or monkey serum possessed therapeutic properties for monkeys inoculated with the virus, whereas normal serum from the same animal sources proved to be inert. Monkeys were inoculated intracerebrally or intranasally with a highly potent virus. Twenty-four hours later treatment was commenced, and the method employed was to inject 2 or 3 c.c. of immune serum through a lumbar puncture needle for three successive days. After an interval of three days more the injections were repeated. The result was that, if the original amount of virus injected was not in excess of a given dose, the infection was either wholly prevented or the onset of the paralysis was much delayed. The power of the immune serums, then, is exercised almost as effectively *in vivo* as *in vitro*. The intraspinal method of administration was selected because lumbar puncture is the most direct route to the central nervous tissues, and the virus in all cases establishes itself in the meninges.

As regards the application of these discoveries to the treatment of the human subject, Netter was the first to treat actual cases of poliomyelitis on these lines, and obtained favourable results in a series of 35 cases. The serum used was from cases of poliomyelitis in which complete recovery from the acute condition had taken place some time previously. The injections were given subdurally as soon as the disease was recognised. The difficulty in procuring the serum had something to do with the determination of the dose, but doses from 5 to 20 c.c. repeated every twenty-four hours are suggested as suitable. Netter found that immune bodies remain in the blood for many years. Nevertheless it is probably advantageous to employ the serum of an individual who has had the disease comparatively recently. Cerebro-spinal fluid should always be withdrawn in suitable quantity before injection. A healthy donor of serum should be selected, and the advantages of a Wassermann test are obvious.

Serum Treatment of Typhus Fever.

Nicolle (*Bulletin de l'Académie de Médecine*, 1st August 1916) has, in collaboration with Blaizot, succeeded in preparing an anti-typhus serum. They had first to overcome the obvious difficulty that the causative micro-organism of typhus is not yet discovered. They therefore prepared emulsions of the organs of guinea-pigs infected with typhus and inoculated them by means of intravenous injection into an ass and a horse. The organs selected were the suprarenal capsules

and spleen, which were regarded as at once highly virulent and the least toxic. The dose for each injection, given every three or four days, was an emulsion of one suprarenal capsule and half of a spleen. Experiments with the serum produced in this way showed that it was useful both in preventing and treating experimental typhus in the guinea-pig.

The results of the treatment of 38 patients are reported. Of these 22 were French, 9 natives of Tunis, and 7 Serbians. The majority were treated by Potel. Only one death occurred, and that in the stage of convalescence from causes quite unconnected with the fever. As two out of three of the first patients in this outbreak treated by Potel had died, the absence of mortality among the serum-treated cases is very remarkable. Great care was taken in verifying the diagnosis in every instance, all the usual bacteriological tests to exclude the typhoid and Mediterranean fevers, relapsing fever and malaria, being always performed. Potel noticed interesting modifications of the temperature. The high continued fever, characterised by absence of remission and maintained at the same level, which is usual in typhus, was either altered in type after the first injection or did not appear at all. Instead, the temperature showed extraordinary oscillations of a kind never seen previously in the disease, and in some instances amounting to 2° Centigrade. In six patients the fever stopped abruptly after the first or second injection, the serum apparently having an abortive action. The mean duration of the 31 cases treated by Potel was 11·6 days, whereas the classical figures of Murchison and others point to a mean duration of at least two if not three days longer. As regards the nervous symptoms so common in typhus, delirium, stupor, and prostration disappeared rapidly or, in cases which received the serum early, did not appear at all. Incontinence of urine and faeces, usually expected in the second week, was only rarely observed. Bedsores were also uncommon.

Potel considers that the serum has a remarkable curative action and is as successful as that employed for cerebro-spinal meningitis. The injections should be given early in the disease, even before the diagnosis is quite certain, and should be repeated daily until defervescence or definite amelioration of symptoms. Improvement is not to be expected before the second week of the fever. The dose should be from 10 to 20 c.c. daily.

Poirson, who treated 7 cases with this serum, agrees with the conclusions of Potel. It will be interesting to see the full reports of the cases, which will be soon published. The results given justify further experiments and, if they are equally successful, Nicolle will have the satisfaction not only of having proved the dissemination of the fever by the louse, but also of having given us a reliable and scientific means of treating the disease. May we hope that it is reserved to him to be the discoverer also of the yet unknown causative micro-organism?

Orchitis and Typhoid Vaccination.

Satre (*Le Progrès Medical*, 5th August 1916) draws attention to an unusual sequela of typhoid vaccination. Several of his patients suffered from congestive reactions of the epididymis, testicle, and cord. The occurrence of orchitis and epididymitis in the course of an enteric fever is well known, although the complication must be regarded as a rare one, and in my own experience it has not occurred more than twice in more than 2000 cases of all ages and both sexes. Olmer and Voisin recently reported some examples and considered that it is not likely to be observed more than once in 200 cases, presumably male adults. But I am not aware of any testicular reaction following anti-typhoid inoculation having been previously described. Satre had noticed some years ago, while inoculating troops in Algeria, that some of the men complained of pain in the region of the cord and testicles, but at that time he did not associate this symptom with the vaccination. In the course of the war, however, he took notes of this complication and is able to report 28 instances of it in a series of 2900 inoculations. He is convinced that there is no actual inflammation. There is swelling and congestion of the glands, which he attributes to modifications of the circulation, which may possibly also cause the diarrhœa and diuresis which are more frequently observed. There does not appear to be any real connection with pre existing venereal disease. It is true that about half his patients had suffered at some time previously from gonorrhœa, and possibly this may in their case have determined a reaction in the organs concerned. But the remaining patients had not suffered from gonorrhœa and, on the other hand, a man convalescing from that infection at the date of inoculation, did not show the reaction. The condition, again, was not related to the rheumatic diathesis. It was quite independent of the severity of the general reaction, that is to say, it was sometimes very slight in cases of a violent general disturbance and, on the other hand, occasionally severe when the general reaction was trivial. Satre remarks that the phenomenon followed inoculation with a triple vaccine more frequently than a dose of any one of the three vaccines of the typhoid group employed singly.

The symptoms usually appeared quickly, on the day of inoculation or the following day. Pain in the groin or actually in the glands was complained of, and this was associated sometimes only with tenderness, sometimes with definite swelling. Occasionally the cord alone was affected, in other cases the epididymis, the testicle, or all three. The condition appears to be always unilateral. The congestion seldom lasted more than three days and never caused any anxiety, although in one of the cases which Satre reports fully as examples there was definite prostration and very acute pain. The treatment recommended is a suspensory bandage. Satre mentions that other medical men of his acquaintance have had a similar experience.

It would be interesting to know if similar observations have been made by British doctors in the course of the vast number of typhoid vaccinations recently performed. No case has come under my own notice, but my experience has not been very extensive. It is possible that the French soldier is more ready to report complications of this sort than is the British, for the latter is undoubtedly very shy on occasion, and the swelling lasts such a short time that he might well find himself better before he had made up his mind to complain. Still, as the question has been raised, it might be well if some medical man who is vaccinating large numbers of troops investigated the subject. The occurrence of orchitis in the actual disease gives the faint suggestion of it in the vaccination reaction a certain interest.

MENTAL DISEASES.

UNDER THE CHARGE OF

JAMES MIDDLEMASS, M.D.

Serological Findings in Paresis and Cerebro-Spinal Syphilis.

Drs. Darling and Newcombe report the results of their investigations of the blood and cerebro-spinal fluid in general paralysis and cerebro-spinal syphilis in patients of the Warren State Hospital (*Amer. Journ. of Insan.*, January 1916, p. 449). Out of 687 admissions 69 were diagnosed clinically as general paralysis, 5 as cerebro-spinal syphilis, and 13 were doubtful. Of the 69 general paralytics, 8 had a negative blood Wassermann reaction, and 3 of the cerebro-spinal syphilities were similarly negative. In all, however, the cerebro-spinal fluid gave a positive reaction. Particulars of these cases are given, as well as details of the technique employed. The conclusions arrived at are:—

1. A positive reaction in the blood seems to be the rule, but even repeated negative findings do not exclude a diagnosis of paresis.

2. The blood Wassermann reaction may or may not be positive in cerebro-spinal syphilis.

3. A positive Wassermann reaction was found in the spinal fluid of all paretics and cerebro-spinal syphilitics.

4. The globulin reaction was very inconstant in both diseases and of no aid in differential diagnosis.

5. The cell count of the cerebro-spinal fluid varied in both conditions, but was as great in one as in the other.

6. The 5 cases of cerebro-spinal syphilis with uniformly positive Wassermann reaction in the spinal fluid, 2 positive and 3 negative bloods, show clearly the difficulty in diagnosis by laboratory methods alone.

7. Two of the cases in which the tests varied at different times demonstrate the necessity of making a series of tests wherever possible.

8. Experience with these and many other cases inclines the authors to believe that all the bloods reported here as negative would have given positive reactions to the Wassermann test if there had been an opportunity to make a series of tests.

9. Satisfactory diagnosis can only be made when the clinical and laboratory findings are considered together.

The Wassermann Test in Practical Psychiatry.

Dr. Lowery of Danvers State Hospital subjected all his new cases to the Wassermann test, and in this paper the results of his experience are given (*Amer. Journ. of Insan.*, April 1916, p. 601). There were 1600 patients, 864 males, 736 females. The test was positive in 19 per cent. of the males, 16 per cent. of females, doubtful in 4 per cent. of both, and negative in 77 and 80 respectively. Of the positive tests 70 per cent. of the males and 35 per cent. of the females were general paralytics. This gives a percentage of 87·5 of positive tests of all general paralytic admissions. In the other forms of mental disease the positive percentage varied from 5 to 10, so that the contrast with the percentage in general paralysis is very striking. The figures show that not in all cases of syphilis in which brain disease develops does that disease take the form of general paralysis. It appears that if a male patient shows a positive test the chances are slightly above even that he is a general paralytic; if a woman, the chance is two to one that she is not. Researches such as this supply a most useful foundation for knowledge of the relation of syphilis to mental disease.

The Treatment of General Paralysis.

Drs. Evans and Thorne in this paper report the results of their treatment of cases of general paralysis by the intraspinous injection of salvarsanised serum (*Amer. Journ. of Insan.*, April 1916, p. 623). The cases treated were 15 in number, selected as a rule because the disease was in an early stage. The method was that known as the Swift-Ellis. After an intravenous injection of salvarsan blood is drawn from the patient and the serum is injected into the cerebro-spinal cavity or into the brain. Their experience of this mode of treatment is not nearly so encouraging as that of Cotton, recently reported in the same journal. They consider that the method is at fault. The quantity of salvarsan injected is so small and so slowly absorbed that its influence cannot be great. The spirochætes that survive the first dose have an opportunity to multiply and continue their destructive work before the next injection. Three of their patients showed improvement both mentally and physically; 5 showed physical improvement only; 2 died

after treatment was discontinued; the remaining 3 showed no improvement at all. One of these, which improved decidedly, died 10 months after treatment was stopped. They consider that as good results can be obtained from the intraspinous as from the intracranial method of injection.

As a contrast to the preceding paper the experience of Dr. Wardner is interesting (*Amer. Journ. of Insan.*, April 1916, p. 643). He treated 14 cases with salvarsanised serum obtained from the patient's own blood, this being injected subdurally. According to his report 5 improved so much as to be able to go back to work and remain at it from 5 to 11 months. One case had a relapse at the end of a year, but responded at once to renewed treatment. Three improved so much as to be able to be placed on parole. Three others are also considerably improved, but are not on parole; 2 have died, and 2 showed no improvement. The operative procedure has not been attended by any untoward complication in the 102 instances in which it was performed. Improvement usually reaches its maximum after six or seven injections. The treatment evidently does not restore the brain to a normal state, as in the two who died the usual changes of general paralysis were found. One died after six injections, and was considered cured five months previously; the other had had four injections and had showed decided physical improvement. Some of his patients had previously had intraspinous injections without benefit.

The Compluetic Reaction in 100 Cases of Amentia.

In this paper Dr. Stephens describes the technique employed, the so-called Wassermann reaction, and the results obtained by it in 100 patients at the Earlswood Asylum (*Brain*, May 1916, p. 27). The term amentia is used in the sense defined by Tredgold, and includes both imbeciles and idiots as well as secondary dements. Of the former there were 76, of the latter 24. Various tables are given showing the results of the tests at different ages, in different diseases, and so on. One of the most valuable is that showing the inherited factors in those which gave a reaction. It would have been still more useful, however, if the same table had included all those in which the reaction was negative, as it is only in this way that the abnormal can be compared with the "average" or standard. As to the reactions, these were in all cases carried out with the blood. The test was found to be definitely positive in 12 cases only: in 16 it was weakly positive, in 14 doubtfully positive. The author states that in his opinion only the first should be accepted as conclusive, but in the tables the other two are included. He also is inclined to the opinion that in all cases the serum should be heated before being tested. As all his patients were males, the relation of sex could not be determined. The reaction was found

in the same percentage of cases of primary as of secondary dementia. It seemed to be stronger and more frequent in idiots than in imbeciles. In all cases the syphilis must have been inherited and not acquired. Sixty two per cent. of reactions occurred in patients between the ages of 12 and 21, but it ought to be stated that all his patients were over 11 years of age. The incidence of the reaction tended to diminish as the age increased. These are the chief conclusions arrived at. The paper closes with a useful summary of the results obtained by others in the same field.

Criminology as a Branch of Medicine.

In this paper (*Med. Journ. of Austral.*, 8th July 1916, p. 19) Dr. Lind puts in a plea for regarding crime and criminals more from the medical point of view than has previously been done. He urges the appointment of specialists in mental disease as full-time officers for the periodic examination of all indicted persons and of all prisoners undergoing punishment. He classifies the various kinds of criminals and analyses the factors which cause crime. Amongst the latter he refers to want of control and discipline amongst adolescents, and the absence of moral influences from State education. The control of literature and picture films should be more stringent, and hooliganism should be prevented by compulsory attendance in evening schools. He has several suggestions regarding the punishment of crime. If looked at more from the point of view of a medical man treating a diseased patient the problem will be approached more with a view to reformation than to vindictive punishment. He is strongly in favour of the indeterminate sentence, and would look on the prison more in the light of a hospital for cure than a place only for detention and punishment. Nearly the same arguments were necessary to bring about the modern transformation of the attitude to asylums, and in time the same or similar results may be obtained in the case of prisons.

A Survey of Defective Delinquents.

In this article Dr. Stearns continues his efforts for dealing more satisfactorily with mental defectives (*Amer. Journ. of Insan.*, January 1916, p. 427). In it he turns attention to the large number of mental defectives who show criminal propensities, and gives statistics referring to the institutions for defectives in the State of Massachusetts. They form about a sixth of the whole. His suggestions are much in the same line as those of Dr. Lind in the preceding review. He is a strong advocate of the indeterminate sentence in youthful criminals, with probationary trial in suitable cases. He also thinks that many patients should be removed from institutions for defectives on account of their

bad moral influence, and that they should be placed in special houses more of the nature of reformatories. This is particularly necessary when the adolescent age has been passed. An Act was passed in the State of Massachusetts in 1911 for dealing with mental defectives, but it has hardly begun to operate yet, apparently from reluctance on the part of the legal authorities to take advantage of it. This shows that public knowledge still lags behind that of medical science, and that the passing of an Act is not equivalent to remedying a defect. As some States in America are fully ten years ahead of this country in recognising the importance of dealing with the problem of the mental defective, experience of the difficulties encountered there should be very valuable when the same problem is resolutely attacked here.

Variations in the Urea in the Fluids of Epileptics.

Professor Obregia and Dr. Urechia have estimated the amount of urea in the blood, cerebro-spinal fluid, and urine of a number of epileptics, and this paper gives particulars of the results (*L'Encéph.*, December 1915, p. 134). Apparently no very definite conclusions can be drawn from the figures. Such as they are the authors state them to be as follows:—In the *blood* the urea is never much increased, the greatest amount found being 0·75 per cent. After a fit there was an increase in 5 cases, in 10 a diminution, and in 2 it was unchanged. Before the fit it was increased in 7 cases, in 2 diminished. In the interval between the seizures there was a slight increase in 3 cases. In the *cerebro-spinal fluid* the quantity of urea is seldom the same as in the blood. It is usually increased before a fit and falls after it. In the *urine* the amount of urea is variable. In 11 it was increased after a seizure, in 6 diminished. Before the fit in 6 cases it was below the average, in 2 above, and in 9 normal. In the intermediate period it was diminished in 21 cases, in 6 increased, in 9 normal. They also carried out estimations of the urea at short intervals after a fit in 8 cases. In 2 of these the excretion was greatly diminished, and in them another fit soon occurred. It is therefore probable that its excretion does not always take place in the usual way when the fits succeed each other frequently, the body being in this way hindered in its attempts to purify itself. In the other 6 cases the elimination of urea was decidedly increased, though the figures show marked irregularity in the curve. These are most apparent from 6 to 8 hours after the seizure. This may serve to explain the discordant results obtained by other workers. A very full bibliography of these previous workers is given.

EDINBURGH MEDICAL JOURNAL.

EDITORIAL NOTES.

WAR HONOURS.

THE Victoria Cross has been awarded to the following two medical officers of the Territorial Forces:—

Captain WILLIAM BARNSLEY ALLEN, M.C., M.B., R.A.M.C.

For most conspicuous bravery and devotion to duty. When gun detachments were unloading H. E. ammunition from waggons which had just come up, the enemy suddenly began to shell the battery position. The first shell fell on one of the limbers, exploded the ammunition, and caused several casualties. Captain Allen saw the occurrence, and at once, with utter disregard of danger, ran straight across the open, under heavy shell fire, commenced dressing the wounded, and undoubtedly by his promptness saved many of them from bleeding to death. He was himself hit four times during the first hour by pieces of shell, one of which fractured two of his ribs, but he never even mentioned this at the time, and coolly went on with his work till the last man was dressed and safely removed.

Captain NOEL GODFREY CHAVASSE, M.C., M.B., R.A.M.C.

For most conspicuous bravery and devotion to duty. During an attack he tended the wounded in the open all day under heavy fire, frequently in view of the enemy. During the ensuing night he searched for wounded on the ground in front of the enemy's lines for four hours. Next day he took one stretcher-bearer to the advanced trenches, and under heavy shell fire carried an urgent case for 500 yards into safety, being wounded in the side by a shell splinter during the journey. The same night he took up a party of twenty volunteers, rescued three wounded men from a shell-hole twenty-five yards from the enemy's trench, buried the bodies of two officers, and collected many identity discs, although fired on by bombs and machine guns.

CASUALTIES.

KILLED in action, Captain CHARLES EDGAR HOLTON SMITH, R.A.M.C., on 16th September 1916.

Captain Smith was educated at Edinburgh University, taking the Scottish Triple Qualification in 1911. He acted as second surgeon of the British Red Crescent hospital in Tripoli in the late Italo-Turkish War, after which he went into practice at Cannock, Staffordshire. He joined the R.A.M.C. on 1st November 1914.

ACCIDENTALLY killed, Captain THOMAS STRAIN, R.A.M.C., while on service, on 16th September 1916.

Captain Strain was educated at St. Mungo's College and Anderson's College, Glasgow, and at the University, where he graduated M.B. and Ch.B. in 1906, and M.D. in 1910. Before the war he was medical officer of Heston and Isleworth Urban District. He took a temporary commission in the R.A.M.C. on 12th April 1915.

KILLED in action, Captain GILBERT VERE BOGLE, New Zealand Medical Corps, on 17th September 1916, aged 32.

Captain Bogle was educated at Edinburgh University, where he graduated M.B. and Ch.B. in 1912, and, after acting as house-surgeon in the gynæcological wards of the Royal Infirmary, Edinburgh, went to New Zealand till he returned to Europe with the Expeditionary Force.

KILLED in action, Captain MAURICE PATERSON INGLIS, R.A.M.C., on 17th September 1916.

Captain Inglis was educated at the Edinburgh University, where he graduated as M.B. and Ch.B. in 1914. He got a commission as lieutenant in the Special Reserve of the R.A.M.C., and was called up for service on 10th November 1914.

DIED of wounds, Major ARTHUR ANDERSON MARTIN, New Zealand Army Medical Corps, on 17th September 1916, aged 40.

Major Martin was educated at Edinburgh University, where he gained the Carlyle Scholarship in Anatomy, the Grierson Scholarship in Materia Medica and Pathology, and the Mackenzie Prize in Practical Anatomy. He graduated M.B. and Ch.B. with honours in 1900. He served as a civil surgeon in the South African Field Force in 1901-2. He returned to this country and graduated M.D. (Edin.) in 1903, taking the diploma of F.R.C.S. (Edin.) in the same year. He then went to New Zealand, where he settled in practice at Palmerston North. He was in this country when the war broke out, and at once joined the R.A.M.C. He was the author of a much-read book on the present war, called *A Surgeon in Khaki*.

DIED on service in Egypt, Colonel BLENMAN BUHOT GRAYFOOT, Bombay Medical Service, on 30th September 1916, aged 56.

Colonel Grayfoot was educated at Edinburgh University, and took the Scottish Triple Qualification in 1885, the M.R.C.S. in 1886, and the M.D. of Durham in 1903. He entered the I.M.S. in 1886, and most of his service was spent in civil employment in Bombay. He went to France in September 1914 as principal medical officer of one of the divisions of the Indian Expeditionary Force. He received the C.B. for his services on 14th January 1916.

DIED on service, Captain DAVID THOMAS CRICHTON FREW, R.A.M.C., on 29th September 1916.

Captain Frew was educated at Glasgow University, where he graduated M.B. and Ch.B., with honours, in 1911. He subsequently took up practice in Glasgow, and in May 1915 took a temporary commission in the R.A.M.C.

KILLED in action, Captain GEORGE MACLEOD LEVACK, R.A.M.C. (T.F.), on 7th October, aged 25.

Captain Levack was educated at Edinburgh University, where he graduated as M.B. and Ch.B. in 1914. He joined the 3rd Highland Field Ambulance in October 1914, and was attached to the Oxford and Bucks Light Infantry when killed.

KILLED in action, Surgeon-Probationer A. L. STRACHAN, R.N.V.R., on 23rd October.

Surgeon-Probationer Strachan entered Marischal College, Aberdeen, as a medical student in the summer of 1913. In his third year he volunteered for service on the call for surgeon-probationers for the Navy, and was appointed to H.M.S. *Genesta*, which was torpedoed and sunk on 23rd October.

KILLED in action, Captain ALFRED THOMAS LOGAN, R.A.M.C., on 16th September, aged 28.

Captain Logan was educated at Glasgow University, where he gained the Dr. Gibson bursary, and graduated M.B. and Ch.B., with distinction in surgery, on 1st April 1915. He was attached to the Grenadier Guards and had recently been mentioned in dispatches.

KILLED in action, Captain GEORGE MOWAT, South African Infantry, on 12th October, aged 43.

Captain Mowat was educated at Glasgow University, where he graduated M.B. and C.M. in 1895. He served as a civil surgeon in the Boer War, and when the present war broke out he joined the Pretoria Regiment, served in it through the German South-West African campaign, and came to Europe with the South African contingent.

DIED of wounds, Captain CHARLES MILL NICOL, R.A.M.C., on 23rd October, aged 28.

Captain Nicol was educated at Glasgow University, where he graduated M.B. and Ch.B. in 1909. He entered the R.A.M.C. as lieutenant on 31st July 1909, and was promoted to captain on 31st January 1913.

British Prisoners of War Book Scheme.

A REPRESENTATIVE committee has been formed to provide British prisoners of war interned in enemy and neutral countries with educational books. We are informed by the secretary of the scheme that there is a great demand from interned medical officers for recent standard works on all branches of medicine and surgery. Readers who may desire to aid in this scheme should send a list of books offered to A. T. Davies, Esq., Board of Education, Whitehall, London, S.W.

Special Graduation Ceremonial.

A SPECIAL graduation ceremonial was held in the M'Ewan Hall on Friday, 13th October, when the Vice-Chancellor conferred degrees in medicine on the following:—

Johannes Jacobus Ackermann, South Africa; Abner Gallie Anderson, Scotland; Ahmed Aziz, Egypt; Arthur Harold de Wet Butler, South Africa; Edward Chapelle, Malta; John Smart Galvin, India; Gerald Wallace Grant (B.A., Dalhousie), Canada (with second-class honours); Balkrishna Rupkrishna Handoo (M.A.), India; Thomas Lavellin Puxley Harries (Surg. Prob., R.N.V.R.), Wales; Frederick John Carlyle Johnstone, Scotland; Aubrey Cecil Kirton, Barbados; John Logan Lamond, Scotland; Charles M'Donald (Surg. Prob., R.N.V.R.), Scotland; Margaret M'Naughton M'Garrity, Scotland; François Hendrik van der Merwe, South Africa; Isabella Morison, Scotland; Godfrey Anthony Paris (Surg. Prob., R.N.V.R.), Malta; Joseph Evelyn Phillips (Surg. Prob., R.N.V.R.), Barbados (with second-class honours); James Ratcliffe, England; Gokal Chand Sahgal, India; Satyendra Nath Seal, India; John Sellar, Scotland; Bernard Hilliard Simon, England.

THE HISTOLOGY OF DISSEMINATED SCLEROSIS.

By JAMES W. DAWSON, M.D., D.Sc.,
Formerly Carnegie Research Fellow.
(From the Royal College of Physicians' Laboratory, Edinburgh.)

Part III.

Pathogenesis and Etiology—A Critical Discussion.

Introduction.
(1) The Nature of the Pathological Process—
1. Developmental.
2. Inflammatory.
(2) Its Origin—
1. In the Neuroglia.
2. In the Nerve Elements.
3. In the Blood-Vessels and Lymphatics.
(3) Etiological Factors—
1. Developmental.
2. External.
(*a*) Trauma.
(*b*) Psychical Shock.
(*c*) Chill.
(*d*) Exogenous Intoxication.
(*e*) Infection and Endogenous Intoxication.
(4) Mode of Action of the Causal Agent—
1. Irregular Distribution and Circumscription.
2. Further Advance of the Process.
3. Modifying Factors.
4. Route of Conveyance to the Nervous Tissues.
Conclusion.

Introduction.

It must be stated at the outset that no satisfactory explanation of the pathological process in all its bearings has been put forward. In the endeavour to interpret the histological observations and to correlate the various conceptions which emerge in the process, it may be possible, however, roughly to estimate the factors which have been at work. It has already been noted that it is necessary to discriminate between the nature of the process underlying disseminated sclerosis and its origin. In its nature it is due either to developmental causes, *i.e.* those inherent in the individual, or to inflammatory causes, *i.e.* those due to infections, intoxications,

circulatory disturbances, etc., these two groups not being necessarily exclusive. In its origin the primary change may arise in any of the constituent elements of the nervous tissue, the neuroglia, the true nervous elements, or the blood-vessels.

For more than a quarter of a century after Charcot gave the first clinical and anatomical picture of the disease, it was looked upon as a distinct morbid entity, a chronic disease in which certain characteristic clinical and anatomical features were always present. Many later writers, however, have related it to a disseminated form of myelitis. They assert that, previously, only the final stages of the disease have been considered, and that, by suitable staining methods, areas may frequently be observed in the same case in all stages of development, from an area of inflammation into an area of sclerosis, from which all traces of inflammation have disappeared.

To this argument it is replied that there exist two forms of the disease—a primary chronic form due to a malformation of the glia, and a secondary myelitic form *en rapport* with infection and intoxication. It is admitted, however, that in primary disseminated sclerosis the development of the focus may begin under the reaction of external factors as *agents provocateurs*, but the disease is in no sense dependent upon these. In opposition to this, it is asserted that the separation into primary and secondary disseminated sclerosis is an artificial and arbitrary one, and that no clinical or anatomical distinction exists between the two. An intermediate position is taken up by those writers who reconcile the inflammatory theory with the developmental one. They admit that congenital anomalies of the glia may become the point of origin of primary proliferations of the glia, but they believe that the blood-vessels distribute some toxi-infectious agent which settles there where the glia is abnormal, and thus calls this glia proliferation into being.

Turning now to the histological observations, it is necessary to ask—Do these throw any light:

1. *Upon the Nature of the Process?*—Do the areas arise solely upon the basis of a gradually increasing glia hyperplasia, or on the basis of an inflammatory reaction, or on both? Further, are there sufficient grounds for distinguishing between the two?

2. *Upon the Origin of the Process?*—If we admit a primary form of disseminated sclerosis of developmental nature, we have answered the question of its origin in the glia; but if we say that the underlying process is of an inflammatory nature, we thereby

also say that the blood-vessels or lymph channels carry the ultimate causal factor to the tissues, but must further decide on which tissue element there is the first evidence of its action.

3. *Upon the Etiological Factors postulated*—chill, trauma, psychic shock, intoxications, infectious diseases, etc.?

4. *Upon the Mode of Action of the Causal Agent*, and the other questions which this consideration involves?

(1) Nature of the Pathological Process.

1. *Developmental.*—Muller has strongly upheld the view of the developmental nature of disseminated sclerosis, and has emphasised the distinction between the primary and secondary forms of the disease. Weigert's work showed that both from the morphological and biological points of view the neuroglia reacts as a true connective tissue. Muller's wider conception of this idea leads him to see that the ultimate product of processes entirely different in their pathogenesis, but presumably all giving rise to focal disease of the parenchyma, may show disseminated sclerotic patches. He divides them, on the one hand, into those processes which always occur as the direct result of exogenous factors and lead to multiple focal degenerations and inflammations, and, on the other hand, into a morbid condition which has one uniform circumscribed pathogenesis, being due not to any known external cause but to a congenital abnormal disposition of the glia. Among the former, he states, are those "comparatively rare" processes which have resulted in injury to the nervous tissue due to some apparently primary blood-vessel condition, *e.g.* syphilitic or arteriosclerotic disease of the vessels, or to a toxi-infective inflammation in the form of a disseminated myelitis or encephalitis. In all these cases the histogenetic terminal product is a secondary disseminated sclerosis. In contrast to these is the "comparatively common" disease caused by congenital disturbances of development, and termed primary disseminated sclerosis. This form, which presents distinct clinical and anatomical signs and may be looked upon as a multiple gliosis (Strumpell), is the only true disseminated sclerosis.

The essential anatomical signs of this condition are the following:—(1) The foci are situated only in those parts of the nervous system which normally contain much glia; and (2) microscopically the foci consist of an excessive proliferation of glia, leading to a dense tissue which is never areolar. The clinical signs are also typical, and Muller thinks that secondary disseminated sclerosis can never successfully simulate Charcot's classical picture in the marked distinctness of each symptom and the peculiarly characteristic course. In spite of the great variableness in the symptoms, which may simulate widely differing diseases of the brain or cord, the diagnosis can always be established by a study

of certain individual symptoms and groups of symptoms, and of the course of the disease. This study will reveal the fact—at first sight paradoxical—that the fundamental features of the clinical condition are in the great majority of cases absolutely the same. There is usually no real exciting cause: it may commence suddenly, but more commonly has a subacute or chronic onset; and it shows sudden exacerbations with marked remissions, but is, on the whole, a progressive and chronic disease.

It is thus seen that the explanation of a "multiple gliosis" is very similar to that given for certain groups of tumours, which must be related to embryonal defects. Ritchie, discussing the etiology of tumours, states that the soundest ground for assigning congenital defect exists when the tumour arises in some part of the body where at some stage of fœtal life the cells of one tissue must push aside those derived from another in order to attain their ultimate natural position: the sequence being that during the building up of the body the cells thus detached or pushed aside, after being embedded for a longer or shorter period, take on a vegetative activity and assume the characters of a tumour. Müller claims that the favourite sites for the development of this gliosis are those which in the embryonal period show specially active processes of growth, or where two surfaces come together and fuse in later development (*Kielstreifen*), or where the marginal zone of the embryonic nervous system is pushed inwards by the ingrowing vessels. At such sites developmental disorders, such as germ invagination or detachment, might set in very readily. The sequence of the process, as has already been pointed out, is a gradually increasing hyperplasia of the abnormal glia in these sites of predilection. This results in a disappearance of the myelin sheath of the nerve fibres, partly through direct compression, and perhaps indirectly by derangement of the circulation.

Areas in secondary disseminated sclerosis, on the other hand, are stated to be of an "areolar" type: there is seldom any sign of real sclerosis; the blood-vessel changes are marked, and the dependence of the areas on the altered vessels is striking. Clinically, also, the disease occurs in definite relation to varied toxi-infective processes: after a shorter or longer period the symptoms either steadily progress or gradually recede to more or less complete recovery; and there is an absence either of any striking fluctuations during its course or of any further relapses.

In all the six cases examined by him Müller found, histologically, a complete absence of the undoubted components of inflammation, and, clinically, in eighty cases, an absence of toxi-infective processes in the anamnesis. He therefore felt justified in excluding exogenous factors and falling back upon developmental causes. In all the nine cases examined by me, none of which, clinically, could be described as "acute

multiple sclerosis," there have been present side by side with dense areas, or forming an outer zone to such, very numerous foci in which transitions could be traced from "early" areas with numerous fat-granule cells—the criterion taken by many writers for the existence of an inflammatory reaction, and which for the present I accept—to areas of almost complete sclerosis in which there was no indication, even at the periphery, of an advancing process. But such complete sclerosis was comparatively rare: even in the most advanced case, many of the areas showed the presence of fat-granule cells in the walls of the vessels both within the area and leading from it, and the great majority of the dense areas showed a central sclerosis, gradually becoming less as it passed into the normal tissue, and this transition zone gave the impression that had time been given it would have undergone an excentric spread comparable to the central gliosis. The end result, then, of all such areas would probably have been a compact gliosis in which were very fine meshes. The examination of many hundreds of areas has satisfied me that "early" areas—so-called areas of fat-granule cell myelitis—can develop into areas of compact gliosis with all the characteristics of those described by Müller as typical of true primary disseminated sclerosis.

From a comparison of the histological study with the above statements it will be seen that I am not in agreement with Müller's view, that the areas in disseminated sclerosis arise solely on the basis of an increasing glia hyperplasia, and that they can be always separated from those arising on the basis of an inflammatory reaction. In this study there is overwhelming evidence that the great majority of the areas have arisen on an inflammatory basis and that a small minority have arisen on the former basis. The end result of both is a tangle of glia fibres. During the process of the gradually increasing glia hyperplasia it is possible, especially in the lateral columns of the cord, to differentiate this mode of formation of a sclerosed area, and I relate such not to a developmental defect of the glia, but to a special reaction in the glia, according to the nature and intensity of the causal factor (see section (4) 3). We have therefore not two affections, or necessarily different stages of the same process, but one causal factor which probably acts with varying intensity.

2. *Inflammation.*—Having therefore given adherence to the inflammatory nature of the process underlying this condition, it must now be considered briefly what is meant by inflammation in the central nervous system.

It is usual to classify the varying histological pictures in the processes termed "inflammatory" in the central nervous system into parenchymatous and interstitial, analogous to the processes in the glandular organs of the body. But the conception of a purely parenchymatous inflammation in the central nervous system has little

anatomical support, for the evidence of changes of a regressive nature in the nerve cells, axis cylinders, and myelin sheaths is accompanied by evidence of progressive phenomena in the other tissue elements. It is not possible, therefore, to draw any clear distinction between simple tissue degeneration and inflammation, and it is usual to designate as "myelitis" both the processes, which are from the beginning distinguished by inflammatory exudations and cell infiltration, and those which begin as degenerations and only in their further course are connected with pathological exudation or proliferative processes.

When, therefore, the inflammatory nature of the process is referred to, it is as a reaction process and one not necessarily associated with exudation of fluid and cell infiltration of the vessel walls. All changes which reveal any kind of progressive phenomena in any of the tissue elements are therefore included in this view of inflammation. Many clinicians have applied the term "acute myelitis" to all diseases of the spinal cord, the symptoms of which cannot be traced to an isolated affection of individual portions of the cord or systems of the cord. Others, before applying the term, make the additional condition that an inflammatory process should really be present. The conditions under which it is justifiable to entitle a disease "myelitis" are not yet defined either clinically or anatomically, and it is the anatomical substratum of the disease, when known, that usually defines the idea of the disease. Bastian, arguing largely from the similarity of the morbid changes to those occurring in the brain, which are due to thrombosis, has long maintained that the great majority of cases are really "thrombotic softening of the cord" rather than an infiltrative myelitis. An ischæmic softened area in the brain, in which great numbers of phagocytic cells, associated later with proliferation of the supporting tissue, are present, is not usually looked upon as an encephalitis. It is the custom to identify this finding by the name of "softening," and yet it is much rather a reactive condition that has set in secondarily, and is a secondary inflammatory reaction—secondary to the presence of the degenerated products. A similar condition may occur in the spinal cord as a result of thrombosis of the spinal vessels, and the reactive phenomena are here again not due to the primary cause which produced the tissue necrosis. On the other hand, as a result of toxi-infective agents, it is possible to get (1) typical cell infiltrations of the vessels and surrounding tissue; (2) simple degenerations of the tissue, in which the so-called inflammatory vascular changes may be absent; or, further, (3) actual inflammatory softenings of the tissue. In the latter two forms we again get later reactive changes in the tissue elements, and these may be secondary to the tissue degeneration, or simultaneously called forth by the primary causal stimulus. Are such conditions to be termed inflammatory only when the primary blood-vessel wall changes are inflammatory? or is it not possible to

recognise the complicated interreaction as an inflammatory process? Clinically, we cannot yet distinguish parenchymatous and infiltrative myelitis, nor these from acute inflammatory softening and acute thrombotic softening. Pathologically, it is sufficient to distinguish two main types in which myelitis may show itself: (1) Infiltration—a form to which some would limit the term "acute" myelitis; (2) softening—to which the term "myelomalacia" has been recently applied—a form which may be very varied in degree from the degeneration of a limited number of nerve fibres, in which the process is more probably subacute, to the necrosis of a large area, in which the process is more likely to be acute in onset.

(2) Its Origin.

It is necessary now to discuss the question of the structural element of the nervous tissue in which this inflammatory process has its origin. In the fully developed sclerotic area changes are evident in relation to the myelin sheath of the nerve fibre, the glia, and the blood-vessels. It is important to recognise on which tissue element the causal agent, circulating presumably in the blood-vessels or lymph sheath of the blood-vessels, first produces its effect.

1. *Changes in the Nervous Tissue.* Charcot defined the histological picture of disseminated sclerosis as a chronic interstitial inflammation leading to a gradually increasing glia hyperplasia. This view is so frequently confused with that formulated by Strumpell, in which there is also a gradually increasing glia hyperplasia, that at the risk of a too frequent repetition the distinction between the two must be emphasised. Strumpell's view is that abnormally placed glia cells take on a latent vegetative activity, and produce a multiple gliosis, while Charcot holds that an exogenous causal factor stimulates the glia anywhere within its range to a marked glia fibril formation. The sequence of the process appeared to him to be the following:—The multiplication of the glia nuclei and concomitant hyperplasia of the reticulated fibrils of the glia constitute the initial fundamental fact and necessary antecedent: the degenerative atrophy of the nerve elements is consecutive and secondary, and the hyperplasia of the vessel walls plays merely an accessory part. The substance of the argument of those who support this view seems to lie in the acknowledged fact that in many areas the glia proliferation is far in excess of that required as a mere reparatory or substitution process, and that it must therefore be looked upon as a productive primary stimulation.

The presence of areas, especially in the lateral columns of the cord, of a gradually increasing glia hyperplasia, has already been indicated, and their comparative rarity in this study emphasised. I am, therefore, not in general agreement with this view, seeing it applies to so few

of the areas, but in isomorphous sclerosis it is probable that the reaction phenomena may be all the more marked when the tissue is not greatly altered, and this would account for the colossal glia proliferation.

A brief allusion may be made to the various functions attributed to the neuroglia. It is no longer held that the glia cells and their processes conduct the nutritive substance from the vessels to the nerve cells and fibres, and it is also probable that the neuroglia must be looked upon as more than a supporting structure or as a tissue element which serves to isolate the ramifications of the neurones. Lugaro has put forward the interesting and suggestive view that the glia serves to transform the products of metabolism of the nervous tissues and to render them inoffensive—the perivascular glia thus acting as a filter. Nageotte and Babes have both ascribed to the glia cells a secretory action in virtue of their derivation from epithelial cells.

2. *Changes in the Nerve Elements—Nerve Fibres and Ganglion Cells.*—Numerous writers have contended that the "noxa" acts directly and primarily upon the nerve fibre before there is any trace of glia proliferation or vessel alteration. This change may be purely degenerative or an actual inflammatory degeneration. The interstitial changes in the glia may be, therefore, on the one hand, secondary to the parenchymatous degeneration, or, on the other, also productive—passing beyond the bounds of a substitution process.

In the histological study it has been pointed out that numerous small areas occur in which there is no appreciable change but a demyelination of the affected tissue. Arguing from the presence of such areas, I am in entire agreement with the view that the most constant and uniform change is the absence of the myelin sheath; this usually commences as an early swelling, varicosity, and faint staining, which passes into a finely granular degeneration.

The glia proliferation, in the great majority of the areas, is, therefore, called forth by two factors: it is immediately occasioned by the stimulant action of the "noxa" which caused the degeneration of the nerve fibre, and it is secondarily brought about by the degenerated products of the parenchyma. The latter effect is explained in part by the irritant action of these products and in part by the well-known conception of Weigert (*Wegfall von Wachstumhinderniss*) that the constituent tissues of an organ are usually in a state of equilibrium, so correlated to one another that no cell can disappear without its place being taken by hyperplasia of the surrounding tissue.

3. *Blood-Vessels and Lymphatics.*—The general agreement of the areas, especially of isolated cerebral areas, with the topographical distribution of the blood-vessels has long led to the belief that these were primarily affected in the diseased process. The changes in the vessels may be expressed, however, in very various ways: a chronic inflammation of the walls of the vessel may affect the nutrition of the

area supplied by it; primary thrombosis or thrombosis secondary to irritation of the intimal lining, by toxins circulating in the blood, might lead to similar malnutrition; some effect, toxic or mechanical, on the intimal lining may lead to minute capillary hæmorrhages; a primary acute vascular change with peri vascular cell infiltration—a true inflammation in the generally accepted sense of the term—might extend to involve the adjoining tissue; or, finally, the vessel wall changes might lead to adhesion and closure of the lymphatic sheaths with a subsequent lymph stasis in the tissues.

During the course of this investigation a large number of small isolated areas, both in the brain and spinal cord, have been cut in serial section with the object of tracing the possible presence of thrombosis or of capillary hæmorrhages. In a few instances, especially in the lateral vessels of the cord and medulla, there have been found aggregations of white cells and the presence of fibrin, which have been taken as indications of intravital thrombosis, but nowhere has evidence been present of organisation of such thrombi nor of alterations in the vessel walls in relation to them, nor have these been always in relation to sclerotic areas. Again, in close relation to the engorgement of the blood-vessels, both within and without the areas, small hæmorrhages have been found. These, however, show no changes, and were looked upon as probably the result of the respiratory difficulties before death. The vessel walls also showed no changes which would explain the hæmorrhages, nor were there any signs of inflammation around them. Keeping in mind the difficulties in recognising small thromboses after actual sclerosis has set in, and also the admitted long time that extravasated blood may remain unchanged in the nervous tissues, it is difficult to account for the origin of sclerotic areas in such primary changes. The absence of any histological evidence of changes in the vessel walls associated with thrombosis or hæmorrhages is quite incompatible with a primary vascular lesion in this sense.

To true inflammatory changes in the walls of the blood-vessels a primary significance has been ascribed by a very large number of writers. It would *a priori* be thought natural that an irritative substance circulating in the blood would, in its filtration and diffusion into the tissues, stimulate the capillary endothelium and cells of the adventitia to proliferate, and that its first effect would thus be on the vessel wall through which it passed. In this investigation special importance was attached to this point, and a reference to the histological study will show that it was impossible to trace, except in rare instances, a primary proliferation of the capillary endothelium, or a primary increase in the nuclear content of the adventitia. The first cell infiltration of the vessel wall was one of fat-granule cells in the adventitial lymph spaces, secondary to the resorptive processes: this called forth a secondary proliferation in the endothelial and other cellular elements

of the adventitia, and at a later stage there was a cell infiltration of lymphocyte-like cells analogous to those found in all chronic processes. In the event, therefore, of a disease-producing agent, either bacterial or toxic, being carried with the blood or circulating in the lymph sheaths, it must be assumed that this poison leaves the blood-channels in so slight an amount or in such weak concentration that a recognisable injury of the vessel wall does not result. The only part played by the blood-vessels would thus be the bringing of the "noxa" to the tissues. The explanation of the numerous findings of such inflammation by other writers must be found either in the presence of areas of an acuter type than were observed by me, or in the possibility that areas were described at a stage when the secondary infiltration of fat-granule cells and the cellular reaction to their presence had occurred.

Alongside of the vessel lesions and dependent on them, disturbances of the lymph circulation have been described. The present writer's views in regard to the changes in the lymphatics and meninges have been stated elsewhere, and it is necessary here only to note that in these observations, in uncomplicated cases, no alteration in the meninges to which any significance could be attached could be found. The chronic inflammation in the vessel walls, with a closure of the lymph spaces, is to be regarded as entirely a late change, so that while I agree with Borst that the fundamental basis of the process underlying disseminated sclerosis may probably be a flooding of the tissues with toxic lymph, I disagree with him regarding the mechanism by which this is brought about in the early stages. In late stages the condensed vessel wall, with adhesion of the adventitial spaces, on its side may contribute to the production of a vicious circle in which hyperlymphosis plays a part.

(3) The Etiological Factors.

In turning now from the nature and origin of the process to its determining cause, we find that there is no positive knowledge of the nature of the agent causing this disease. The etiological factors postulated may be discussed under two groups—developmental and external.

1. *Developmental.*—In the discussion of the pathogenesis it was shown that Müller based his view overwhelmingly on the presence of an endogenous (developmental) process, under which naturally lay endogenous factors—inborn defects. One weak point in his arguments, however, lies in the fact that the evidence of congenital anomalies in the anamnesis is hard to find, or completely absent. Groups of cases occurring in families also have only been reported in one or two rare instances, and in this connection it must be remembered how great is the difficulty in the differential diagnosis between disseminated sclerosis and certain atypical forms of hereditary disease; and, further, that such

cases might be family diseases with a symptom-complex like that of disseminated sclerosis. Again, cases of congenital multiple sclerosis are even more isolated. A neuropathic disposition has been alleged as an important etiological factor, but this association has not been substantiated, and where present no causal significance has usually been ascribed to it. Most observers note that patients were normal both physically and mentally.

Furstner and others speak of "an early invalidity of the central nervous system," which is unloosed by later accidental factors. It is evident that such views do not bring the solution of the problem much nearer, yet it must be admitted that in some cases "congenital anomalies of development may be present in the sense that they lay the foundation of the constitutional tendency which renders the individual more susceptible to the injurious influences of later life" (Mott). Such anomalies may be insufficient to give rise to any symptoms till the onset of these later factors, but they indicate a diminished resistance or a diminished vitality of the nerve elements such as Gowers postulated for Friedreich's ataxia ("abiotrophy").

2. *External Factors.*—Among the more immediate factors must be considered the following: trauma (physical shock), psychical shocks, chills, infections, and intoxications. Modern conceptions of disease do not admit of primary importance being attached to the influence of cold, injury, strain, and emotion in the production of diseases of the nervous system, but such influences, recent or remote, are repeatedly brought into relation to the onset of such diseases. As a rule we cannot go beyond the *post hoc ergo propter hoc* argument, for there are no other data available, yet it is necessary to consider briefly whether the histological data throw any light upon the mode of action of such influences, which are at least admitted to bring about a lowered resistance of the nervous system to the causal agent.

(a) *Trauma.*—Slight degrees of dystocic lesions of the infantile spinal cord may lead frequently to slight degrees of hæmorrhage, and it is thought not improbable that these may be the basis of the diseases of the spinal cord which are related to the formation of multiple areas of sclerosis, or to the formation of cavities and later syringomyelia. Mendel is one of the principal advocates of the traumatic etiology of disseminated sclerosis. He believes that as a result of the trauma an impulse of movement is transmitted to the cerebro-spinal fluid, that this leads to pressure effects in the most delicate channels of both brain and spinal cord, and that through these alterations of pressure there may occur minute ruptures (of capillaries) and hæmorrhages. From the assumption that small hæmorrhages are sufficient to evoke symptoms of disease, there justifiably follows, therefore, the view that in the case of cerebral and spinal "commotio," intra-medullary hæmorrhages are responsible for the symptoms. That the symptoms frequently pass

away rapidly must be ascribed to a rapid resorption of the poured-out blood, but it may be argued that, since permanent disturbances frequently remain after shock, processes of degeneration in the nerve elements, which are beyond our present methods of recognition, have occurred. Hence the expression "molecular alteration of the nerve elements." The slighter degrees may lead to transient symptoms, the severer to focal areas of degeneration, which thus have their rise in a molecular alteration resulting from "commotio."

The assumption of a molecular alteration of the nerve elements is hypothetical, since it does not rest on demonstrated microscopic findings at the time, but in the opinion of many writers intramedullary hæmorrhages or outpourings of lymph into the tissue, of traumatic origin, may be followed by swelling and degeneration which lay the foundation of a *locus minoris resistentiæ* for toxic infective agents. It is also thought possible that in such damaged areas, still capable of functioning, the damage may be completed by any other factors which lower the resistance of the tissue. In relation to multiple hæmorrhages, it is relevant to mention the view put forward by Taylor and others, that at least a portion of the spinal cord degeneration found in some cases of pernicious anæmia is due to primary focal degenerations caused by hæmorrhages similar to those which occur in the retina and elsewhere in this disease.

(b) *Psychical Shock.*—The influence of severe terror and other marked emotions has often been related not only to the onset of the disease, but to a relapse or the aggravation of a present condition. A possible explanation may be found in the sudden changes of the circulatory conditions in the nervous system occasioned by such influences as act specially on the vasomotor system. The profound influence of the vasomotor system in the production of disease has scarcely been recognised, and in a later section its possible mode of action in disseminated sclerosis will be considered.

(c) *Chills.*—Krafft-Ebing has attributed considerable etiological significance to chills. In forty out of a hundred cases he traced this factor, and represents the relationship between the two in the contraction of the vessels favouring ischæmic degeneration of nerve fibres in localised areas—especially if collateral anastomoses were absent.

In regard to cold, fatigue, and psychical influences, it is probable that these act as immediate factors by lowering the vitality or the resistance of the individual, or in allowing, through vasomotor influences, the emergence of a toxin already circulating in the blood. In the case of trauma, the possibility of the actual rupture of capillaries or of lymphatics must be admitted, and the consequent hæmorrhages or the toxic lymph thus admitted in sufficient concentration into the tissues will effect the primary degeneration of the nerve fibres and a secondary proliferation of glia.

(d) *Exogenous Intoxications.*—Oppenheim has laid special stress on the influence of lead, copper, and zinc, also of alcohol and carbonic oxide, but it is evident that some more general causal agent must be at work, although such intoxications may be important as predisposing factors in the production of a toxic arteritis.

(e) *Infectious and Endogenous Intoxications.*—Toxi-infective agents have long been held of primary importance. Pierre Marie has urged the recognition of acute infective disease as the actual final cause of disseminated sclerosis, which he believes arises on the basis of multiple inflammatory vessel alterations brought about by the infective agents of scarlet fever, measles, diphtheria, smallpox, puerperal fever, pneumonia, erysipelas, typhoid fever, cholera, etc., in combination with the organisms which produce a mixed infection. No organisms, however, that can have any causal relationship to the disease have ever been identified, either in the blood, the cerebro-spinal fluid, or the tissues.

Tubercle and syphilis are both generally admitted to have no significance in the etiology. Syphilis may produce disseminated areas in the central nervous system, but the histological characters of these have, as a rule, nothing in common with those of disseminated sclerosis, in which disease also the reactions in the serum and in the cerebro-spinal fluid and the cytological examination of the latter are all negative.

Endogenous intoxications are of a very varied nature, and include not only the toxins engendered within the body by bacteria, but also auto-toxins caused through abnormal metabolic or assimilative processes, and those the result of deficient or abnormal secretions. Systematic investigations of metabolic changes in disseminated sclerosis have not yet been undertaken, nor has any toxin of any nature been isolated either from the blood or the cerebro-spinal fluid. Investigations of the biochemical changes, analogous to those found in parasyphilitic affections, have thrown little light on the disease, but investigations along this line have not been extensive.

It is the unsatisfactory and contradictory result of each of the above-mentioned factors that has led numerous writers to come to the conclusion that few of the alleged causal factors are of any importance, and to seek for one common cause which will explain the real nature of the disease. Müller, from the large number of causes postulated, has drawn the inference that none of these can be the essential one, and therefore feels justified in falling back on his view of a developmental cause to which any of the other factors may be an exciting agent. It has already been pointed out that the assumption of the developmental genesis of the disease is insufficiently founded, and such a conception must appear, in the light of modern pathological knowledge,

untenable, and justifiable only when every other factor has been eliminated.

The essential problem, then, lies still before us—What is the nature of the special stimulus which originates this process? In the attempt to answer this question it is important to remember that the causal factor must be in operation over long stretches of years, allowing remissions of the disease and relapses. It would, therefore, appear necessary to exclude those agents which actually attack the organism and then disappear rapidly. It is admitted by all, except the supporters of the developmental nature of the process, that the topographical distribution of the areas points to the blood-vessels or their lymphatic sheaths as the route of conveyance of this agent, and the assumption of an infection or intoxication harmonises with this relation to the blood-vessels.

The forms of myelitis have been classified by Taylor and Buzzard as infective, toxic, and syphilitic. The last-named variety shows changes which are readily recognised as quite distinct from the changes in disseminated sclerosis, so that it need not be further considered. Inflammatory vascular changes, with cell infiltration, may be regarded as the primary reaction of the nervous system to the virus of poliomyelitis, and cases of acute infective infiltrative myelitis, with organisms present in the foci, have been described by Purves Stewart and others. Again, the experimental work of Marinesco and others has shown that areas of infective and toxic myelitis may be produced by a number of bacteria and bacterial toxins, and a comparison of the histological changes in the two forms with those of similar conditions in man gives the following as the general differential histological characteristics. In infective myelitis we have dilated and engorged vessels, with an excess of nuclear elements in the adventitial sheath, probably derived from the proliferation of the structural elements of the adventitia rather than from the blood (Taylor and Buzzard). Later, there is proliferation of the glia elements and changes in the nerve cells and fibres, partly due to œdema and to the vascular changes, and partly to the toxic products of the invading organisms. In toxic myelitis, on the other hand, the changes are much less marked: the patchy areas of degeneration are unaccompanied at first by any cell infiltration of the vessel-adventitia or of the tissue, and there is, as a rule, marked swelling and varicosity of the axis cylinder and myelin sheath. The later reactive changes in the glia depend on the intensity of the primary change.

From the evidence of an infiltrative poliomyelitis caused by the virus of poliomyelitis, from the recorded cases of acute infiltrative myelitis in man, and from the experimental production of infiltrative myelitis by bacterial injection into animals, it is natural to assume that organismal infection is more often associated with primary inflammatory (proliferative) changes in the blood-vessel walls. Taylor and Buzzard,

however, point out that some organisms may at one time and under certain circumstances determine an infiltrative form of myelitis and at other times a toxic, and that we are not fully acquainted with the laws which govern such different results. The balance of evidence, however, suggests that an infiltrative myelitis is usually due to an organismal cause, and most workers conclude that areas of necrosis, without cell infiltration and independent of vascular thrombosis, must be ascribed to toxic influences. The toxic myelitis of pregnancy may be given as an evidence of the result of an auto-intoxication which is often responsible at the same time for serious trouble in connection with the cardiac and renal functions.

Turning now to the histological data previously given, it will be recalled that the conviction has been expressed that nowhere was there evidence of a primary vascular inflammation, but that the stimulus acts primarily on the myelin, and almost simultaneously on the glia cells, surrounding the blood-vessels. The changes which have been outlined in a normally developing "early" area are thus much more nearly allied to those of a toxic myelitis than those of an infective myelitis, and differ from the former only in degree.

In the absence, however, of any definite proof of such a toxin either in the blood or cerebro-spinal fluid, other data must be submitted with great reserve, and, in spite of the absence of any positive evidence for the presence of bacteria, the organismal cause of disseminated sclerosis cannot be excluded, especially in view of the conditions existing in syphilitic and parasyphilitic affections on the one hand, and poliomyelitis on the other. The example of general paralysis, in which disease Noguchi has demonstrated the presence of the spirochæta pallida in the cortex, but which formerly was ascribed to a chronic toxic encephalitis of lymphatic origin, must serve to show how quickly views regarding the pathogenesis and etiology of a disease may require to be modified.

The important point, however, in relation to disseminated sclerosis is that experimental work has proved that circumscribed areas may be attacked by toxic materials, and that in such areas the first effect is on the nerve fibres and interstitial tissue surrounding the blood-vessels, and only secondarily on the vessel walls themselves. The causal agent must be a weak one compared to the usual agents which produce toxic myelitis, for there is evidence of a slow, often limited and localised action, and the subacute onset in the relapses again argues for its slight intensity. For such a chronic course as disseminated sclerosis usually runs, with many remissions, the "noxa" responsible must remain constant in the body, lead at certain periods to relapse, or even remain latent for a long period. The assumption of an auto-toxin agrees with this demand, and the example of pernicious anæmia in which the remissions may possibly be traced to the result of absorption into the

portal tract of some poisonous substances may serve as a possible analogy. The assumption of an organismal stimulus moving about in the body or malignantly stored in depôts such as the spleen or bone-marrow, or of the elaboration products of such organisms continually reforming and leading to new symptoms, would also agree with this demand. It is impossible to say that this is improbable, for the relapses in malaria and relapsing fever coincide with the reappearance in the circulating blood of free sporocytes of the malarial organism or of the spirillum of Obermeyer, respectively: such relapse is therefore a reinfection from within. In parasyphilis, also, it must be supposed that the specific organism in some form enters the central nervous system, remaining latent for years either in the lymph vessels or in the brain tissues proper, and exerting its effect either by its direct action or by its toxins. The influence of such factors as infectious diseases, cold, trauma, etc., would thus be to produce an impairment of the nervous tissues, and so predispose them to degeneration from auto-toxins and other causes; or the lowered body resistance would allow of sufficient concentration of the toxin, through deficient elimination; or a combination of both factors, impairment of the tissues and an increasing intensity of the "noxa," probably acts in varying degrees in every case.

The views that have been put forward as to the source of the possible "auto-toxin" can be only briefly touched upon. M'Cormac suggests that disseminated sclerosis is due to physico-chemical causes, which may be in operation for a long time, and thinks that as indol, skatol, and phenol—toxic products of intestinal putrefaction—might produce structural changes in the liver which are followed by nervous symptoms, it is possible that actual structural changes might occur in the nervous system itself. Bramwell suggests several possibilities: the toxic agent may be the result of deranged metabolism, possibly in the liver or some other organ, or it may be generated in and absorbed from the gastro-intestinal tract, as is probable in pernicious anæmia. The effect of exposure to cold in the production of hæmoglobinuria is explained by the probable rapid production of some toxic substance, the result of deranged metabolism. It is thought that the exposure to cold acts reflexly through the nervous system on some central organ—perhaps the liver—and produces a poison which is the cause of the rapid destruction of red blood-cells, and that disseminated sclerosis might be the result of a chemical poison similarly formed. Another possibility is that the agent may not be a toxin properly so called, but that the composition of the blood is so altered that some substance or substances necessary for the nutrition of the nervous tissues, especially the nerve fibres, is absent, or that the blood contains some substance which acts injuriously on the nutrition of the nerve fibres. Dixon Mann thinks that disseminated sclerosis is due to an

unknown autogenous toxin, which probably acts by setting up changes in the ultimate vascular supply of the part affected.

(4) The Mode of Action of the Causal Agent.

The principal questions which present themselves in this section may be stated thus:—

1. Why and how is the process so irregularly distributed and at first circumscribed?
2. What is the cause of its further advance?
3. What factors cause modifications in its mode of action?
4. By what route, blood or lymph channels, is it conveyed to the nervous tissues?

1. *Its Irregular Distribution and Circumscription.*—If we could trace the disease to the entry of corpuscular elements, such as bacteria, or thrombi infected by such, or emboli, there would be no further difficulty in accounting for circumscribed vascular areas being irregularly attacked. If it is not, however, a question of the immediate action of bacteria at an identical point, but probably of toxins circulating in the blood, how is it that these agents in solution in the blood choose such irregularly distributed spots?

From the frequent presence of a central large vessel in the area it has been assumed that this vessel, by some alteration in its walls, limited to a certain definite stretch of its longitudinal course, was responsible for the sclerotic area. It is difficult to understand how toxins would diffuse from such a vessel, instead of following the capillary area of ramification, and it is probable, as has been frequently shown in the histological study, that it is not a question of one central vessel, but of many transverse and oblique vessels, giving the impression that not one single vessel, but the branches of a blood-vessel stem are the starting-point of the process. Serial sections have frequently given definite proof, as the area was followed up, that it broke up into individually distinct smaller areas, which were related to the component branches of the previous central vessel. These sections, again, showed the gradual fusion of such areas around the ramifications of a vessel, and marginal strands of myelinated fibres still left between each primary focus.

This view, however, removes only a portion of the difficulty. If certain areas of distribution, large or small, are affected, what factors determine the exact areas chosen? For this no satisfactory explanation has been suggested, but certain analogies may be given. In peri-axial neuritis in man, the result of chronic lead-poisoning, not only is the radial nerve picked out, but certain discontinuous areas are primarily affected in a manner very closely resembling the discontinuous myelin sheath degeneration in disseminated sclerosis. Gombault and Stranksy

have also experimentally produced similar discontinuous areas in sub-acute lead-poisoning in animals. In these cases the toxin circulating in the blood is known to affect only certain irregularly distributed areas. The selective action of toxi-infective agents on particular parts of the nervous system may be further illustrated by the action of the virus of syphilis and that of rabies: by the effect of arsenic and that of the neurotoxin formed by the diphtheria bacillus, on certain nerves; and, further, by the tetanus toxin, which, like strychnine, acts mainly on the synapses of the reflex arcs of the brain stem and spinal cord, reducing their resistance. Further, in the combined degenerations found in pernicious anæmia, cancerous cachexia, etc., the toxin postulated must choose out certain definite vessel territories, and the symmetry may be explained by the two halves of the cord being exposed to the same diffusely acting agent.

The sequence of events in disseminated sclerosis is probably that in the circulating blood the "noxa" escapes from the capillaries and transition vessels, and acts upon the tissues in the sense that the area of supply of the vessel is affected, *i.e.* it passes over into the tissues with the nutritive fluid. Williamson believes that the cells of the capillary walls act as true secreting cells, and when they are stimulated there is an increased flow of lymph into the surrounding tissue. Certain substances, such as toxins produced by micro-organisms, have the power of stimulating the endothelial walls, and as the endothelial walls of the central nervous system of capillaries and lymphatics are extremely delicate and active, the lymph flow is great. Numerous writers suggest that the toxins produce an unrecognisable injury of the vessel wall by which it becomes more permeable, and thus allows of the increased transudation of the lymph. Others think it possible that a transient paralytic dilatation of the vessels permits an exudation, which in other conditions does not pass through the vessel wall, or is arrested by the glia filter. From their recent experiments, of placing bacteria-laden capsules in the abdominal cavity of rabbits, Orr and Rows are of opinion that the influence of the sympathetic cannot be excluded, and that the involvement of this mechanism here and there causes dilatation and stasis in certain parts of the vessels, favours the formation of hyaline thrombosis, and hence myelin atrophy and sclerotic areas. The conditions of the circulation in the central nervous system are so little understood, and the vasomotor mechanism so complicated, that such an irregular and localised vasomotor action may be quite possible.

None of these suggestions explain why certain areas are chosen while adjoining areas of distribution are, at least primarily, unaffected, and it may be that its causation has appeared unnecessarily mysterious, and that it is quite analogous to the irregular distribution of other processes, *e.g.* the haphazard distribution of areas in experi-

mental toxic myelitis, or the atheromatous patches on vessel walls. It cannot be supposed that the tissues are in a state of proportionate equilibrium or equal resistance, and that there are not varying degrees of oscillation, and pressure, and permeability in the vessels. The filtration from the vessels, including whatever part secretory processes may play in the passage of the lymph through the vessel walls, may thus be related to alterations, the nature of which are as yet little understood, but which have a place in the normal mechanism of the body.

2. *Further Advance of the Process.*—The ebb and flow of the symptoms are, clinically, amongst the most characteristic features of the disease, and, anatomically, "early" areas are the expression of this recurrence of a long-standing disease. When the primary direct effect of the causal agent has become exhausted the areas become sclerosed, and in such cases there may be a true remission; but frequently, in addition to "early" areas, there are found at the periphery of the old areas indications of an advancing process. This may be due to the persistence of the action of the primary agent, or to the fact that the products of degeneration had caused a secondary reaction after the infective or toxic agent had ceased to work, or, possibly, to a new process surrounding the older one. Such recent areas show that the morbid agent persists in the organism. Muller has urged that the similarity of the basal features of the clinical and anatomical pictures points to one common cause of the disease. He thinks that we cannot assume that, for example, the toxi-infective agents of the acute infectious diseases, *i.e.* of so different a nature, will always lead to one and the same definitely characteristic disease-picture. A similar view has been upheld by numerous writers, and, in the absence of any specific organismal or toxic cause, an explanation has been sought for along very varied lines. Redlich thinks it possible that the characteristic symptom-complex is caused by the "general functional injury" to the nervous system; that the acute infectious disease, whose cause we otherwise look upon as specific, may yet produce a definite metabolic disturbance, which, in its action upon the central nervous system, is specific. Marburg also looks upon disseminated sclerosis as a "meta-infectious" disease in this sense. Such a term seems justifiable in view of the use of the term meta- or para-syphilis, in which it was believed that the syphilitic virus induced in the body profound metabolic changes. Mott has pointed out that these resulted in a large amount of lipoids occurring in the serum and in the cerebro-spinal fluid; that these same lipoids are found in the normal body fluids, so that the specific character is thus manifested by quantity rather than by quality. The relations of syphilis to the parasyphilitic affections are of special interest in relation to the possibility that a latent organism may be the cause of disseminated sclerosis. Before Noguchi's discovery of the spirochæta

pallida in the cortex, Mott and Sewell had defined these relations thus—(1) The syphilitic organism is in itself the causal organism; (2) there is produced within the body as the result of syphilis a toxin, and this post-syphilitic toxin is the cause; or (3) syphilis produces an impairment of the nervous system and so predisposes it to degeneration from auto-toxins and other causes. It is in this final sense we look upon Redlich's and Marburg's conception—that acute infectious diseases of very varied etiology may produce once for all a specific impairment of the nervous system, and so predispose it to degeneration from auto-toxins. It has also been suggested that the original agent may cause, in addition to the evident areas of degeneration, alterations in other portions, and that these later, not from a persistence in the organism of the causal agent, but from excess of function, strain, or circulatory disturbances, may degenerate. Again, that the presence of degenerative processes in the central nervous system may suffice in themselves for the production of further areas of degeneration. For either of these views there is no foundation or analogy. In the absence, therefore, of any positive evidence as to the determining factor, it can only be presumed that relapses are due either to the intermittent evolution of a toxin, or to an unknown organism which, in the interval between a remission and a relapse, lies latent and inactive. The causation of pernicious anæmia, as we have seen, is thought to be due to the similar intermittent evolution of a toxin, and the discovery of the spirochæta pallida in the cortex in general paralysis is a sufficient analogy for the latter alternative. Mott has suggested that this organism may exist in a latent, granular, or intracellular form in the parenchyma of the nervous system, where it cannot be reached by drugs such as arsenic, mercury, and antimony.

3. *The Factors which cause a Modification in the Action of the Causal Agent.*—The variations in the histological picture have been ascribed by different writers to two main causes—the fact that the process was observed at different stages, and to the different nature of the etiological factor in individual cases. Apart from such basal differences, and from the variations due to the influence of complications, there are modifications in the development of the process which can be attributed only to the fact that its progress, assuming one common causal factor, is not always typical, uniform, and progressive.

Ainley Walker, in discussing the relative changes in the tissue elements in inflamed areas, points out that stimulation and injurious irritation differ only in degree, and that whatever is capable of causing injury will, if sufficiently diminished in intensity, exert a stimulant action. On the other hand, stimulation, if sufficiently intensified or long maintained, becomes irritation and produces injury. He further points out that one and the same agency may have a different effect on different types of cells, or even on different cells of the same type. The

changes which occur in the tissues of inflamed areas are, therefore, of two types—degenerative and regenerative respectively. Both of these occur simultaneously in different orders of cells, *e.g.* the parenchyma of an organ may undergo degenerative change while its connective tissue basis is proliferating. If we apply this to the changes in disseminated sclerosis, we can conclude that "there are certain principles underlying the inflammatory process which enable us to recognise in it different degrees of one process rather than several independent series of reactions." This shows that a common cause, according to the intensity and duration of its action, may produce a very varying picture.

It is probable that when the causal agent diffuses through the blood vessel walls with average concentration and intensity, areas arise on the basis of (1) a primary solution of the myelin—a result of the irritant action of the "noxa"; and (2) a simultaneous glia reaction—the result of the stimulant action of the "noxa," and that such areas pass through stages of fat granule cell formation, glia fibril formation, to a complete sclerosis with very fine, dense glia meshes. On the other hand, if the causal agent be of weaker concentration and intensity and acting over a longer time, its stimulant action on the glia is solely in exercise; areas would then arise on the basis of a gradually increasing glia hyperplasia. The comparative picture presented by the end-result of each could be differentiated only by the possibly more isomorphous character of the resultant sclerosis in the latter case. Further, the "noxa" may be of stronger intensity and its irritant action predominate, so that areas of a more acute myelitic character arise, in which the rapid degeneration of the myelin sheath is accompanied by a destruction, not a mere swelling, of the axis cylinder. Such areas, followed by a certain amount of secondary degeneration, and later by a reparatory sclerosis, are also found in disseminated sclerosis, but this is not the normal evolution of an area. At the same time it is clear that a varying intensity of the "noxa" might cause areas to arise in which the resultant sclerosis shows all transitions from a simple retention of the glia network—the so called "areoliette" areas, through areas with a certain but not complete degree of fibril formation—in which the proliferated glia nuclei are the nodal points of radiating fibrils to areas of complete sclerosis in which the glia meshes are very close.

It is difficult to answer the question whether an acute encephalomyelitis can pass over into a disseminated sclerosis. The pathological conceptions of the disease, as have been indicated, pass over into one another, and with this conception is admitted the inflammatory nature of the process. Whether it can be regarded as inflammation, as degenerative inflammation, or as purely degenerative, depends fundamentally upon different definitions of the same process. It seems defensible to regard certain forms of disseminated sclerosis, especially those occurring in close relation to the acute infectious diseases, as

having their origin in an acute disseminated encephalo-myelitis; but, in spite of the seeming inconstancy of the symptoms and the irregular incidence in the position of the areas, it seems justifiable to regard disseminated sclerosis as an inflammatory process with a subacute onset and slow progressive course—often distinguished by remissions and acute or subacute relapses which depend upon the development of new areas, and to regard the final cause as a true specific "noxa," which may be either a metabolic disturbance or a special infective stimulus.

4. *Route of Conveyance of the Causal Agent to the Tissues.*—Writers admit two paths of infection of the central nervous system: the one —which has been looked on as the more constant—the blood-stream, and the other the lymph stream. The possibility of lymphogenous infection of the nervous system has received much attention since experimental evidence was established in favour of the spread of rabies and tetanus by the lymph channels of the nerves. Its increasing recognition in this country is largely due to the work of Orr and Rows, who have taken as the principle of their research the fact, demonstrated by experiments with organisms and coloured fluids, that the lymph stream in peripheral nerves is an ascending one and capable of conveying infection to the central nervous system. The lymphatic path within the cord has, in the main, an outward direction, as is demonstrated by the presence of fat-granule cells, containing the degenerated products of the nervous tissue within the adventitial spaces; but numerous experiments and histological observations leave no doubt that it admits of a current inwards—thus admitting an invasion by cellular elements, micro-organisms, and toxic substances.

The experiments of Orr and Rows tend to prove that infection passing into the cord by the lymphatic system takes a definite course, that the structures of the cord and the nerves react to infection by this path in a definite manner, and that the inflammation can be propagated by the toxic lymph to parts distant from the focus of greatest intensity. It is recognised that when once the inflammatory condition has been established within the spinal cord, the toxic lymph spreads by direct continuity, and this continuity of extension is looked upon as characteristic of lymphogenous infections. Numerous clinical data, from cases in which peripheral inflammatory foci existed, have been brought forward, in support of this experimental work, to demonstrate the facility with which infection spreads along the lymph sheaths of nerves to the spinal cord.

In relation to acute poliomyelitis it may be stated that experimental evidence proves without doubt that it can be produced by lymphogenous infection, and the view that the virus may enter by means of the lymphatics and thus exert its first effect upon the meninges is strengthened by the anatomical findings. The lymph spaces surrounding the vessels of the cord are, anatomically, processes of the arachnoid

space, and the lymph in them is in connection with the cerebro-spinal fluid.

It has been assumed by most writers that the causal agent in disseminated sclerosis circulates in the blood, but the possible lymphogenous source of the infection has been supported on the following grounds—(1) The endovascular changes are much less marked than the perivascular; and (2) the pathogenic significance of the periventricular sclerosis.

This localisation was such a striking feature in several of the cases that early in the investigation it was recognised that ependymal and peri-ependymal lesions lead to important considerations in reference to the toxicity of the cerebro-spinal fluid. Bullock's recent experiments point to this toxicity, and Lhermitte and Guccione found that when carmine was injected into the lateral ventricles of a dog, it was found, ten days later, almost wholly in the sheath of the sub-ependymal veins and in the sub-ependymal tissue. It is logical, therefore, to assume that toxi-infective agents in the cerebro-spinal fluid might follow the same route. The absence, however, of any change in the ependymal epithelium seemed to contra-indicate the possibility of a simple soakage of the cerebro-spinal fluid into the periventricular tissue and also to contra-indicate the irritating character of the fluid, for a granular condition of the ventricular walls is usually associated with such a change in the fluid. The terminal branches of the central arteries ramify on the ventricular walls, and this localisation of the areas, therefore, might equally be related to the vascular richness of this region. The cell infiltration and proliferation around the sub-ependymal veins, which constitute the path of return of the periventricular circulation, would then again be simply an indication of the resorptive processes consequent on myelin degeneration and not a result of a peri-ependymitis.

Conclusion.

In the preceding critical discussion of the questions that come into consideration in a study of disseminated sclerosis it is made clear that no complete interpretation of the subject can be given and that imperfectly known factors have to be suggested for the solution of several of the problems. The tentative nature of the explanations on many points, and the fact that no uniform conception of the process can be offered, have, therefore, been taken into account in formulating the following conclusions:—

(1.) That the process underlying disseminated sclerosis is a subacute disseminated encephalo-myelitis, which terminates in areas of actual and complete sclerosis.

The histological study has given evidence that the great majority

of the areas arise on the basis of an evolution through a stage of fat-granule cell formation, and this conclusion has been reached on the ground, therefore—(1) Of the nature of this evolution; (2) that acutely inflamed areas are rarely present; and (3) that in subacute processes the general architecture of the tissue is retained.

(ii.) That there are no sufficient grounds for distinguishing between primary and secondary forms of disseminated sclerosis.

A small minority of the areas had evolved through an increasing glia hyperplasia, but this varying picture, seen best in glia-stained sections, depends not on different underlying processes, but more probably on the varying intensity of one causal agent, and the varying factors which modify its action in individual cases and at individual times.

(iii.) That there is much to favour the view that true disseminated sclerosis is due to a specific morbid agent which calls forth a clearly defined clinical and anatomical picture; that other disseminated affections of the central nervous system, such as disseminated arteriosclerotic, syphilitic, endarteritic, and acute encephalo-myelitic processes, may all produce a symptom-complex very similar to that of disseminated sclerosis, but that they, in their further course, differ from the latter in the characteristic remissions and relapses; and, further, that acute infective diseases, trauma, chill, shock, and all known exogenous factors may act as exciting factors in lowering the resistance of the organism, and thus allowing the final determining factor to operate.

The most important form of these disseminated affections is that due to toxi-infective conditions, which may give rise to disseminated areas of myelo-encephalis. It seems unlikely that the specific agents of the acute infective diseases can each produce a clinical and pathological condition so characteristic as that of disseminated sclerosis, while it is conceivable—and we know from experimental and other evidence that it is possible—that the various toxi-infective agents can call forth an acute disseminated myelitis which runs its course and remains stationary. The clinical symptoms would resemble disseminated sclerosis, but the characteristic remissions and relapses would be wanting. The pathological and clinical concepts of the two diseases, therefore, pass into each other; the differential diagnosis must rest, clinically, on the further course of the disease, and, anatomically, on data which differ only in degree.

The grounds for this conclusion are based, therefore, largely upon an increasing knowledge of the selective character of toxins. Several instances of such have been given in an earlier section, and here

allusion may be made to progressive lenticular degeneration—a symmetrical degeneration of the lenticular nuclei—which Wilson traces to the selective action of the morbid agent on these collections of grey matter. Analogies can never be conclusive, but they at least mark the possible direction in which we may look for a solution and allow of the suggestion that a characteristic anatomical and clinical picture is called forth by a specific morbid agent.

(iv.) That the causal agent is of the nature of a soluble toxin, and that it is conveyed to the nervous tissues probably by the blood-channel rather than by the lymphatics.

It has already been frequently stated that the nature of this postulated specific morbid agent which is the final and determining factor in the process is quite uncertain, and that there is not sufficient evidence to show whether it is microbial or of the nature of a toxin. There is much in favour of the view that the disease is toxic in origin; the histological evidence, which is far from conclusive, is related to the absence of cell infiltration of the vessel walls, for it is admitted by most writers that areas of degeneration without cell infiltration are more usually due to toxic influences, while infective agents more uniformly call forth an infiltrative form of myelitis. More decisive, however, is the clinical evidence; many of the early symptoms—symptoms which sometimes last for years—are slight motor palsies and transient psychical symptoms, which pass off readily under treatment with faradism or suggestion—such transient symptoms are very suggestive of the persistence in the body of some toxin which exercises its action on the nervous system. The variability of the early symptoms, which has so frequently led to the diagnosis of hysteria, suggests that at this stage areas may be affected by a "nutritional" rather than a structural change. The possibility of dynamic modification of function is now well recognised in diseases of the nervous system, while as yet structural change is slight, and this being so, it is therefore more likely that the agent producing this varying disturbance of function is some form of toxin. The importance of determining the relation of the so-called Westphal-Strumpell pseudo-sclerosis to disseminated sclerosis is very great, for in this affection the symptoms of disseminated sclerosis are present with negative pathological findings.

(v.) That the restriction and distribution of the pathological process is in some way related to the selective action of the toxin on certain areas of the blood-supply, or that unknown factors determine an irregularly distributed paralytic dilatation, with an increased filtration through the vessel walls.

The difficulty of explaining how the degeneration connects itself

with special vascular tracts, and avoids others, has led many writers to fall back upon the assumption of developmental defects or minimal tissue injuries, which form *loci minoris resistentiæ* where the circulating toxin might settle. In the histological study it has been pointed out that it is not a question of one central vessel, a portion of the longitudinal course of which is affected, but rather of a blood-vessel stem with its terminal branches or a division of its branches. It is probable that the circulating toxin escapes from the capillaries and transition vessels in the sense that the area of supply of the ramifications is affected. It has been suggested that the toxin may have a chemiotactic influence on certain portions of brain tissue, but it is difficult to conceive of such an irregularly and widely distributed chemiotaxis unrelated to definite functional nuclei or fibre-systems. The toxin may, however, conceivably select certain areas of blood-supply, for it is known experimentally that certain substances can influence the blood-supply of regional parts of the brain ; and Meynert has suggested that this may be the explanation of the maniac-depressive psychoses, which he looks upon as neuroses of the vasomotor system limited to certain cerebral areas. The recent experiments of Orr and Rows also point to the possibility of a localised affection of the vasomotor system due to the action of toxins on the sympathetic nerves. In the present state of our knowledge, as it is impossible to determine the final causal agent, it is equally impossible to determine the factor which allows of its diffusion through certain terminal areas of the ramification of a blood-vessel.

(vi.) That remissions and relapses necessitate the assumption of the latent presence of the morbid agent in the body, or, if this is an autogenous toxin, either its intermittent evolution or its accumulation from deficient elimination.

Analogies have been found in certain diseases, both microbial and toxic in origin, in which relatively the same exacerbations and remissions occur. Allowing that it is microbial in origin, we may find an analogy in cerebro-spinal syphilis in which there are also the same exacerbations and remissions. If we admit, rather, its toxic origin, the closest analogy is probably found in pernicious anæmia, where it is thought likely that both the hæmolysis and the cord changes are due to the same toxin. The source of the toxin is here also uncertain, but the fact that gastro intestinal disturbances are so common in this disease lends support to the view that it is of intestinal origin, and that its intermittent evolution or its insufficient elimination lead to the remissions. Such analogies can, again, not be final, and in the latter case the parallel breaks down from its incompleteness. There is no known association of disseminated sclerosis with any changes in the gastro-intestinal tract or elsewhere : the connection, if any, with glands

of internal secretion has never been worked out; the general metabolic changes occurring in the body have not been investigated; and the cerebro-spinal fluid is known not to exhibit the cytological and biochemical changes which are so marked in parasyphilitic affections. The investigations of the cerebro-spinal fluid have as yet been very incomplete, and the evidence, which tends to prove that it is toxic, does not decide whether it is so as a result of the process or whether this toxicity is its cause.

(vii.) It is suggested:

(*a*) That fleeting early motor paralyses and psychic symptoms may be related to the presence of areas in association paths; that remission of these symptoms is possibly due to the linking up of other association paths, or their compensation by the opening up of new paths; and, further, that the variation of the symptoms in all stages of the disease emphasises the importance of the mental factor in the symptomatology.

It is a little difficult to explain the mechanism of the fleeting early palsies and psychical symptoms. The possibility of an early dynamic modification of function has already been noted, but the period now referred to is one when presumably structural changes have set in. I consider that the involvement of the psychic areas at such a stage is to be explained in the same way as the affection of the motor areas. In such cases it is possible that the motor area or tract involvement is insignificant, for, if it were the essential lesion, the recovery could never be so rapid. Every movement requires the integrity of a large number of association areas, but we realise this only when we watch a child trying to walk, or a paralysed person trying to move. It is possible, therefore, that in these transient palsies the disturbance is in one of the association paths. With each recurrence of an area in such paths, motor images and memories would be progressively blotted out till the motor area is so cut off from its usual associations that progressive paralysis occurs. So long as an axis cylinder remains a stimulus is capable of passing. The destruction of the myelin sheath probably results in the increase of the resistance to the passage of any stimulus. If a sufficient number of association areas can be linked up around a particular function, in order to elicit from it a discharge sufficient to overcome this resistance, then remission is possible. By stimulation through suggestion, faradisation, etc., these remissions may be brought about with a success dependent upon the degree to which the paralysis is psychic or motor. In this the extra stimulus is probably gained by the linking up of distantly connected association areas and thus utilising or deviating their energy to the desired process. It is also possible that when one path is blocked by disease, others, perhaps not so direct, or possibly new paths, may be opened up.

(*b*) That the cortical areas share in the production of psychical symptoms: but, probably, when these are defined they are dependent upon the inhibition of the action of those cortical cells which control the thalamic centres—the latter being intimately related to the emotions and forming part of Langley's autonomic system. Such inhibition would take place by the presence of subcortical areas in relation to the cortico-petal fibres and to the presence of areas in the optic thalamus itself. Reference has already been made to the possibility that early psychical symptoms, such as restlessness, emotionalism, involuntary fits of laughter, may, together with the early transient palsies, be referred to a functional change which precedes an anatomical, structural change.

The changes in the cortex have been held to account for the psychical symptoms, often so marked a feature in the clinical picture. Such symptoms, however, have been present in cases which anatomically showed little cortical change; and, again, marked involvement of the cortex has been noted in cases which clinically presented no change in the intellectual or psychical functions.

The essential change is a demyelination, and the cyto-architecture of the cortex is frequently retained. On the other hand, the ganglion cells may show a marked increase of their satellite cells or all stages of degeneration, but these changes are not strictly limited to the area of demyelination. The glia cells in the deepest layers are markedly proliferated and show all stages of glia fibril formation; in the layer of the deep pyramids there is an hypertrophy of the normal glia cell content with formation of fibrils—insufficient to lead to sclerosis: in the upper layers the glia cell changes diminish in intensity, but fine glia cell forms with long processes of uniform calibre are present, especially around the ganglion cells and capillaries; and in the subpial marginal layer there is again an increase of both cells and fibrils. Cortical and subcortical areas were numerous in six of the cases, few in two, and in the ninth case not evident—but this brain was the only one not examined microscopically in large sections.

(*c*) That volitional tremor, alterations in speech, and nystagmus, which are all largely due to want of co-ordination, may be the result of sclerosis spreading in from the several ventricles and the aqueduct.

The distribution of the areas lends strong support to the view which associates tremor with disturbance of the afferent and efferent extra-pyramidal paths. These, according to Kinnier Wilson, are respectively the cerebello-rubro-thalamo-cortical path from the nucleus dentatus of the cerebellum by the superior cerebellar peduncle to the

nucleus of the opposite side, and thence to the inferior and external divisions of the optic thalamus, and so to the sensory and motor cortex. The efferent path is the lenticulo rubro-spinal system, by the ansa lenticularis and subthalamic region to the red nucleus, and thence by the rubro-spinal tract of Monakow to the anterior horns of the spinal cord. Lesions of the former path probably remove the inhibitory function of the cortico petal fibres which pass to the cortical cells, and lesions of the latter remove the normal inhibitory or steadying influence which the corpus striatum exercises on the anterior horn cell. The proximity of the structures innovating the eye muscles to the aqueduct and the angles and floor of the fourth ventricle accounts for the nystagmus and visual symptoms. That these paths are all specially liable to be affected is well brought out in nearly all the cases. (The early eye symptoms which are so frequently the first sign of the disease are probably the result of areas in the optic radiations as they pass near the descending horn of the lateral ventricles; the later and more serious symptoms to areas in the optic tracts, chiasma, and nerves.)

(*f*) That as the lesions are disseminated through the cerebro-spinal axis the disturbances are, therefore, especially liable to affect systems which are extensive.

Thus the pyramidal fibres, in their long course from the cerebral cortex to the anterior horn cells in the cord, are invariably and usually early affected. This affection may at first be so slight as to be evidenced only in a unilateral loss or diminution of the abdominal reflexes, or it may be so severe as to give a spasticity great enough to abolish the deep reflexes. As the co-ordinating system has peripheral, spinal, vestibular, cerebellar, and cerebral components, it is co-extensive with the nervous system: some part of this extensive system is almost invariably implicated. Nystagmus, tremor—both static and intentional—and ataxia all occur: the spasticity to a certain extent masks the inco-ordination in the gait and gives rise to a spastic cerebellar type of progression. In spite of the length of the afferent paths of common sensation, loss of common sensibility is rare. The cause of this anomaly is uncertain, but it is possible that a considerable degree of involvement of the sensory columns may occur without a marked loss of sensibility, and that afferent sensory stimuli survive when weaker efferent stimuli fail.

It has been already pointed out that many of the symptoms are due to loss of cerebral inhibition, and this loss of cerebral control is a source of great confusion in interpreting the symptoms. Just as the interception of the volitional motor impulses permits increased irritation of the reflex mechanism, so does the loss of the unconscious

cerebral control permit increased irritation of the automatic, the sympathetic, and the reflex mechanism.

The frequent presence of daily variations in the symptoms after the disease has become established emphasises the importance of the mental factor in the symptomatology. Structural lesions do not thus vary, but the external evidences of them—the patient's reaction to them—may vary. This variation probably depends upon environmental stress, upon nutritive factors, and probably, chiefly, upon the intermittent evolution of endogenous toxins.

We are now in a position to turn to the conclusions based on definite histological data.

(viii.) The "areolar" zones, the true "areolar" areas, and the perivascular sieve-like areas must probably be referred to an œdema of the perifocal and perivascular tissue, due to alterations in the blood and lymph circulation, firstly within the area, and then in the general tissue—through the presence of numerous foci. This is a secondary process, and is the result and not the cause of the sclerotic areas.

Areas of "shadow" sclerosis which have a definite outline or surround, with such an outline true sclerotic areas must be ascribed to a diffusion of the toxic lymph in concentration insufficient to cause a complete demyelination. Such areas are often accompanied by a diffuse glia hyperplasia.

Further, areas of diffuse and lighter staining, which connect true sclerotic areas—sometimes over long stretches of tissue—may be caused by several factors, among which may be mentioned—(1) A diffuse spread of the toxic lymph in the lymph spaces; (2) circulatory derangements in consequence of numerous foci; (3) considerable participation of the axis cylinders causing a certain degree of secondary degeneration.

Finally, especially in the cord, there are frequent evidences of an early degeneration of the myelin sheath (Marchi staining), over the whole myelinated tissue and the nerve roots. This must be referred to the general somatic disturbances.

(ix.) Sites of predilection are probably related (1) to the vessels: to the terminal ramifications of end-arteries, *e.g.* on the ventricular surfaces, and to the points where vessels break up, *e.g.* in the transition zone between grey and white matter—both in the central and cortical grey matter; and (2) to areas where much glia is normally present; again, therefore, to the periventricular and to the pericentral tissue, to the optic chiasma, to the postero-median and para-median septa, to the marginal glia zone, and to the perivascular glia layer.

The periventricular affection was very marked in six of the cases, slight in other two, and scarcely noticeable in one. The optic chiasma and one or both optic nerves were affected in seven out of the eight cases in which they were examined, and in six cases there was an extensive involvement of the optic radiations on both sides—an involvement which seemed to extend laterally and posteriorly from the sclerosis around the posterior horn of the lateral ventricle.

The frequent marked symmetry of the areas may be related to both of these circumstances.

(x.) *Ganglion Cells.*—The ganglion cells in the grey matter of the spinal cord, and in the analogous nuclei in the medulla oblongata and pons, retain for a long time, even in the advancing sclerosis, their nucleus and chromophile granules. There is considerable histological evidence to show that this accounts for the absence of secondary degeneration in the anterior nerve roots, and, in part, for the remission of the symptoms—for there is no reason to suppose that such cells do not function.

The later varied changes in the ganglion cells must be ascribed to (1) the increasing condensation of the sclerotic process; (2) the absence of function; and (3) the associated somatic disturbances—the latter two factors will also influence the cells throughout the non-sclerotic tissue. I have never seen in the cord an increase of the satellite cells such as has just been described around the cortical ganglion cells.

(xi.) *Axis Cylinders.*—The persistence of numerous axis cylinders in the sclerotic areas has been accepted as an axiom by most writers, and with this view I am in entire agreement. Secondary degeneration is therefore not well defined, but it affects a certain number of fibres.

In the early areas the axis cylinders undergo a swelling, which may go on to a granular disintegration, but those that survive the swelling, or have not shared it, persist in the advancing sclerosis for a long time. This circumstance has been held to explain (1) the absence of secondary degeneration; (2) the remissions: the gradual retrogression of the symptoms must be related to the resorption of the fat-granule cells, the swollen axis cylinders then diminish in volume, and impulses would thus be able to proceed by means of the denuded axis cylinders—giving, therefore, a remission of the symptoms; and (3) the intention tremor. It is thought that this impulse would be carried on irregularly in a broken or jerky manner and would thus produce the oscillations which disturb the due execution of the voluntary movements. It is supposed that this is effected in part by the absence of the insulating myelin sheath, which allows of the diffusion of the

impulse to neighbouring axis cylinders and also by the pressure of the increasing glia. It must be remembered, however, that though function is related in development to the presence of the myelin sheath, there is, as far as we know, no evidence that non-medullated axis cylinders do not transmit impulses as regularly as medullated fibres. Again, fibres lying adjacent to each other in an area do not end necessarily at the same level or in adjacent cells. The leakage, then, if it existed, would take place from the entire circumference, and the result would be not an inco-ordinated act, but a very feeble one, or none at all. Further, the glia meshes in which the naked axis cylinders lie must themselves insulate the fibre.

(xii.) *Myelin Sheath of the Nerve Fibre.*—The change in the myelin sheath must be looked upon as the most constant, the most uniform, and in many cases the primary one. This has all the characters of a primary degeneration in contrast to those of a secondary degeneration, and is due to the destructive or irritant action of the stimulus. The myelin sheath is presumed to have an insulating action; it probably also facilitates the transit of nervous impulses.

(xiii.) *Neuroglia.*—The glia changes set in, as a rule, simultaneously with those in the myelin sheath. The earlier reaction is due to the stimulant action of the morbid agent; and the later reaction is secondary also to the degenerative processes.

The glia changes correspond to the age of the process and its intensity—on the one hand glia cell proliferation, with the formation of large protoplasmic, potential fibril forming cells, and later, glia fibril formation at the expense of this protoplasm. Glia cells which have produced fibrils undergo slow, regressive changes; the glia nuclei subsequently atrophy and disappear, and to such a disappearance is due the fact that the old sclerotic areas have fewer nuclei than the normal tissue. The course and direction of the glia fibrils is, as a rule, in the direction of the normal longitudinal course of the nerve fibres, especially in posterior columns of the cord, but frequent whorls are found, or a dense tangle with very fine meshes.

In the progressive alteration in the glia all its component parts share—nucleus, protoplasm, and specific fibres. The "glia limitans perivascularis" is the first to show signs of reaction. The abundance of the glia in an area justifies the name "sclerosis"; but the process may stop short of complete sclerosis, and so areas are present showing all degrees of density of the glia meshes. The sclerotic tissue is more loosely constructed in the grey matter of the cord and the analogous nuclei.

The glia changes in the cortex lend support to the view that the

adult glia consists of (1) cells, with nuclei, cell bodies and protoplasmic processes; (2) differentiated fibrils, whether anatomically independent or not; (3) intercellular fibreless glia.

It seems unnecessary to postulate areas of glia abnormality and that the blood vessels carry the morbid agent to such points; but this can neither be proved nor disproved.

(XIV.) *Blood-Vessels.*—The topographical relation of the areas points to the blood-vessels, or their lymphatic sheaths, as the route by which the "noxa" is conveyed to the tissues. In its diffusion through the vessel walls the morbid agent causes no recognisable primary alteration, but probably there is an abnormal permeability and diminished resistance to the oscillations of the blood-pressure by which an increased transudation of (toxic) lymph is made possible. The only anatomical expression of this is dilatation and engorgement.

On the ground of numerous serial sections of areas it has been concluded that it is the branches of one vessel stem which are affected, and that the primary minute areas, related to each branch, subsequently coalesce.

There is no evidence of a primary nuclear increase in the endothelium or in the adventitia. The first cellular increase is secondary to the resorptive processes—an infiltration of fat-granule cells. This is followed by a proliferation in all the cell elements of the adventitia, and, at a later stage, by a modified infiltration of lymphocyte like cells and a few plasma cells. The derivation of the nuclear content of the adventitia at a later stage is, therefore, a very varied one. The later changes in the vessel walls are related to a condensation of its adventitia, such as is found in all chronic conditions. The separate layers of the adventitia gradually blend, its nuclear contents break up, and are carried away in the lymph spaces or fuse with the adventitia; and, finally, both adventitia and media show a homogeneous "hyaline" change in which nothing can be recognised of specific muscle, connective tissue, and elastic elements.

(XV.) *Fat-Granule Cells* (*Fettkörnchenzellen*).—The first fat-granule cells probably arise from the proliferation of the small round glia cells, and, at a later stage, also from a proliferation of the endothelial elements of the adventitia.

They absorb the degenerated myelin in the form possibly of a solution, which is precipitated as granules in the protoplasmic substance of the cell. They pass, or are drawn in by suction and the pressure of the increasing glia fibrils, into the lymphatic sheaths of the capillaries

and transition vessels, and thence by the larger vessels to the inner layers of the pia. On their way large numbers are broken up *in situ*, and their crenated nuclei can for a long time be recognised as one of the nuclear elements of the adventitia.

(xvi.) The cranial and spinal nerve roots are frequently involved in the glious portion of their extramedullary course.

Such changes in the posterior spinal and analogous nerve roots may account for the existence of the sensory changes, which are almost always present to a slight extent, and especially for the trigeminal neuralgia.

(xvii.) *The Meninges.*—The occasional variations in the meninges are related to complications and are probably of no significance in the pathogenesis of the disease.

Approximately final answers can, therefore, be given to the questions relating to the nature of the process underlying disseminated sclerosis, to its origin, to the relation of several secondary etiological factors, and to certain aspects of the mode of action of the final causal agent. We are, however, still quite in the dark concerning the nature of this final cause which determines, anatomically, a process so well defined, and one without any close analogy; and, clinically, a disease which, however variable the early symptoms, conceals its characteristic course only temporarily. The frequent presence of the cardinal symptoms, in spite of the seeming irregularity and incidence in the position of the areas and their restriction, seems to point to a certain constancy in the changes, the nature of which is not yet fully known, but which may be dependent on the production of a specific metabolic disturbance, due to a latent organism or an auto-toxin. All that is most important, therefore, still remains for future investigations along bacteriological, serological, and experimental lines, which have recently done so much to clear up the etiology of other affections of the central nervous system.

CLINICAL STUDIES. XI.—VERY MARKED SPASTIC PARAPLEGIA OF FIFTEEN YEARS' DURATION; DEATH DUE TO DIABETES MELLITUS AND BOILS. DURING THE WHOLE COURSE OF THE CASE NO SYMPTOMS OR SIGNS OF DISSEMINATED SCLEROSIS. POST-MORTEM FINDINGS SHOWED ALL THE CHARACTERISTIC FEATURES OF DISSEMINATED SCLEROSIS IN THE SPINAL CORD, PONS, AND MEDULLA—THE ONLY PARTS OF THE NERVOUS SYSTEM WHICH COULD BE EXAMINED.

By BYROM BRAMWELL, M.D., F.R.C.P.E., LL.D., F.R.S.E.,

WITH

REPORT ON THE EXAMINATION OF THE SPINAL CORD.

By JAMES W. DAWSON, M.D.

Everyone who has had much experience of disseminated sclerosis knows that the disease not infrequently begins with symptoms indicative of spastic or spastic-ataxic paraplegia, and that it may be only after some years that the characteristic features of disseminated sclerosis (such as nystagmus, intention tremor, partial optic atrophy, speech affection, etc.) are developed. Many cases of this kind have come under my own observation; consequently, in every case of spastic paraplegia for which there is no apparent cause, *i.e.*, in which the spastic paraplegia appears to be primary, more particularly when the disease occurs in a young woman, I suspect the probable subsequent development of the characteristic symptoms of disseminated sclerosis. In most of these cases in which spastic paraplegia is the first symptom of disseminated sclerosis, the spasticity and the paraplegia are comparatively slight in degree.

Cases of spastic paraplegia with most marked rigidity and complete inability to walk, the legs being kept stiff in extension, are occasionally seen in which there is no obvious cause for the spinal lesion (sclerosis of the crossed pyramidal tracts). In these cases one is led by exclusion to diagnose *primary* spastic paraplegia—a disease which probably occurs, but which is extremely rare, so rare that a diagnosis of *primary* spastic paraplegia should always be distrusted.

In the following case, which was carefully observed for seven

years, most marked spastic paraplegia was the only condition which was present. The patient was again and again examined in the hope of detecting some of the characteristic symptoms of disseminated sclerosis. Such symptoms never developed; but post-mortem examination showed the characteristic lesions in the spinal cord of disseminated sclerosis. Consequently the case is one of quite exceptional interest and rarity.

CLINICAL REPORT.

By BYROM BRAMWELL, M.D.

FEMALE, aged 45, single, seen 26th November 1906, suffering from most marked spastic paraplegia, apparently primary.

History.—The patient enjoyed good health until six years ago, when, after sleeping in a bed with damp sheets, she commenced to suffer from weakness and stiffness in the legs. She is very definite that sleeping in the damp bed was the cause of her illness; before that she always enjoyed good health.

Condition on Examination.—General health very good. A woman of great mental ability and intellectual development. Very marked spastic paraplegia—lower extremities very stiff and kept in an extended position: able to walk with very great difficulty. Muscularity in the legs good. Suffers every now and again from pain in the lower part of the back, and occasionally has a girdle sensation. Latterly has had some difficulty in urination—precipitate urination, some forcing, occasionally dribbling. Bowels very constipated. No evidence of transverse myelitis; no signs of disseminated sclerosis; no sensory disturbances; no giddiness, no headache, no defective eyesight; no volitional tremor; no optic atrophy. Both knee-jerks very markedly exaggerated; double ankle-clonus; double Babinski. Abdominal reflexes absent. No visceral disease.

Diagnosis.—Spastic paraplegia for which there is no apparent cause: (?) primary spastic paraplegia. The possibility of the case being one of disseminated sclerosis was entertained, but there were absolutely no symptoms or signs, except the spastic paraplegia, of that condition.

Subsequent Progress of the Case.—The patient was repeatedly seen, and subsequently examined during the years 1906 to 1911. On more than one occasion she was seen by my son, Dr. Edwin. The spastic paraplegia continued extremely marked in degree, the legs being kept stiffly extended and the patient unable to walk. With the exception of the rigidity, weakness in the legs and alteration in the reflexes described above, no other symptoms developed. The symptoms and signs of disseminated sclerosis were again and again carefully looked for with a negative result. The general and mental health remained good.

On *24th May* 1913 the patient was admitted to Chalmers Hospital, suffering from spastic paraplegia and diabetes mellitus, and was discharged from hospital on 29th July 1913. During her stay in hospital the amount of sugar passed *per diem* varied from 700 to 1300 grains.

On *13th April* 1915 she was readmitted to Chalmers Hospital, suffering from spastic paraplegia, diabetes mellitus, and three large boils on the head—one over the forehead and two on the back of the neck. She died with symptoms of acetone poisoning on 11th April 1916.

The spinal cord, pons, and medulla were removed post mortem by Dr. A. M. Drennan (permission to make a complete examination was refused). The microscopical examination was subsequently made by Dr. J. W. Dawson, whose report follows, and shows that the lesions in the spinal cord were typically those of disseminated sclerosis.

PATHOLOGICAL REPORT.

By JAMES W. DAWSON, M.D.

(From the Royal College of Physicians Laboratory, Edinburgh.)

MACROSCOPICALLY, there was no thickening of the dura mater or other membrane of the spinal cord. On section of the cervical and dorsal cord at various levels the lateral columns showed a greyish gelatinous degeneration while other segments appeared normal.

Microscopically, the most extensive affection of the cord was found in the cervical enlargement, in the lower dorsal and third lumbar segments; in other sections the whole transverse section of the cord stained normally. The medulla oblongata and pons were comparatively slightly affected, and there was no involvement of the floor and lateral walls of the fourth ventricle. The brain was not examined.

At several levels of the cord, especially in the sections taken from C5, C8, and from D8, D10, there was a marked symmetry in the position and form of the areas present in the right lateral column—a symmetry which was continued throughout the segments and gave almost the impression of an ascending and descending secondary degeneration. It is to be noted, however, that the sclerosis at any one level passes over the boundaries of a system degeneration; and, further, that the examination of sections taken from segments between these levels, *e.g.* from C7 to D9, shows clearly that the sclerosis, quite defined in one segment, is indicated in the adjacent one by diffuse and faint staining. Such areas of "shadow" sclerosis seem to unite areas of complete sclerosis, and these changes must be related to a commencing secondary degeneration.

The general histological characters of the areas correspond closely to those usually found in cases of disseminated sclerosis. The central zone, showing a complete absence of myelin sheath staining, is occupied by a dense mass of neuroglia fibrils with small glia nuclei and a few

persistent axis cylinders. Around the periphery of some of the areas there is a zone, indicative of an ascending process in which numerous fat-granule cells are present in Marchi sections. A few areas were found, chiefly in the lower dorsal cord, in which the process is altogether at an early stage, and Marchi sections showed vessels radiating to the surface of the cord, with their lymphatic sheaths filled with fat-granule cells, while similar cells were scattered abundantly throughout the intervening tissue. The changes in the blood-vessels in the older areas were those related to a condensation of their adventitia, such as is found in all chronic conditions. The alterations in the ganglion cells, similarly, were those associated with a slow sclerotic process or with absence of function. Numerous axis cylinders could be recognised in the sclerosed areas, especially in those in the right lateral column, but many of these persisting axis cylinders were swollen and altered in staining, changes which must be related to altered function.

Topographical Distribution of the Areas—Spinal Cord; Cervical Region.—In sections at the level of C1 a small area of sclerosis was present at the tip of the right anterior horn, involving the fibres of the bundle of Helweg. At C3 there is an indication in the form of "shadow" sclerosis of an area in the right lateral column. C5 shows—(*a*) A very advanced large patch in the right lateral column. The only normally staining fibres in this column form a band, varying in width on the surface of the cord; the sclerosed tissue involves also the antero-lateral and postero-lateral angles of grey matter. (*b*) A small patch in the anterior root zone, separated from (*a*) by practically normal fibres. (*c*) Partial degeneration in the right posterior columns, both of Goll and Burdach. (*d*) Partial degeneration in the left lateral column in the position of the crossed pyramidal tract. (*e*) A patch of partial degeneration in the left anterior root zone symmetrical with the patch (*b*) in the right lateral column (see Fig. 1). At C6 the whole of the right lateral column and grey matter is demyelinated, leaving intact, however, the reflex collateral fibres of the posterior roots as they enter the posterior horn, and a band of peripheral fibres corresponding to the dorsal and ventral cerebellar tracts. An ill-defined area of complete sclerosis, verging into "shadow" sclerosis, is also present in the right posterior columns, together with a peripheral zone of partial degeneration in the anterior part of the left lateral column (see Fig. 2). At C8 (upper part) the sclerosis in the right lateral and posterior columns is confined to small areas in the centre of extensive "shadow" sclerosis, while in the lower part of this segment the posterior columns stain normally; but the demyelination of the lateral columns has extended not only to the surface of the cord, but across the right anterior horn, and involves the anterior columns of the same and opposite sides.

Dorsal Region.—The upper four dorsal segments are quite normal;

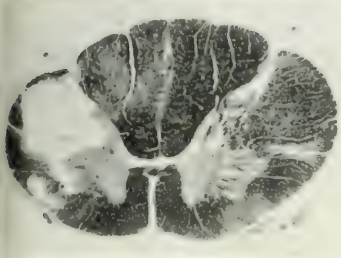

FIG. 1.—C5. Weigert-Pal.

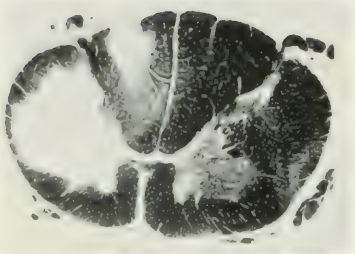

FIG. 2.—C6. Weigert-Pal.

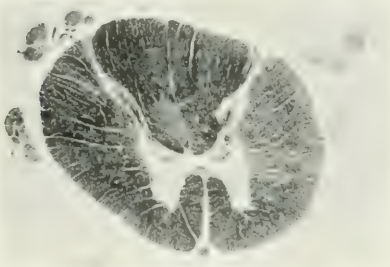

FIG. 3.—Upper Dorsal. Weigert-Pal.

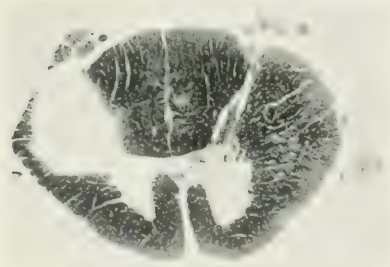

FIG. 4.—D8. Weigert-Pal.

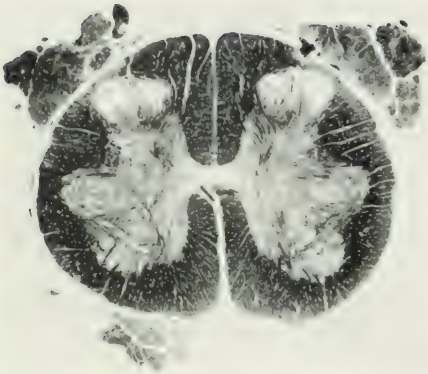

Fig. 5.—L.L. Weigert-Pal.

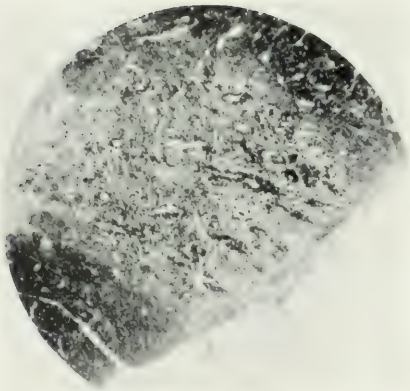

Fig. 6.—D.S. Marchi.

there is no lesion in any of the tracts (see Fig. 3). In D5 one large oval area occupied the whole of both right and left posterior columns, leaving a zone of normal fibres peripherally; a second small area was present immediately anterior to the substantia gelatinosa Rolandi and tract of Lissauer. From D5 to D10 the sclerosis, complete or "shadow" in character, was almost uniform, and affected the right lateral column anterior to a line drawn outwards from the postero-lateral horn of grey matter (see Fig. 4). At D11 the right posterior horn was surrounded on either side by a zone of "shadow" sclerosis, and an early area was present in the anterior portion of the left lateral column, extending from grey matter to periphery of cord (see Fig. 6).

Lumbar Region.—The only segment affected is L3. Sections at this level show (*a*) a well-defined advanced patch in right lateral column in front of the crossed pyramidal tract; this sclerosis involves also the antero-lateral and postero-lateral groups of anterior horn cells; (*b*) patch of partial degeneration in left lateral column symmetrical with (*a*); (*c*) and (*d*) patches of slight degeneration in each direct pyramidal tract. The posterior fibres of the lateral columns on both sides also show a degree of lesser staining. L4 was normal (see Fig. 5).

Medulla Oblongata.—Sections through the decussation of the anterior pyramids show a single lateral patch which involves the ventral and dorsal cerebellar tracts, the substantia gelatinosa Rolandi, together with a few of the fibres of the ascending fifth nerve. In sections through the upper part of the medulla oblongata and cerebellum the sclerosis is confined to the medullary cores of the vermis; it does not involve the roof nuclei nor the white matter of the cerebellum. As we ascend the brain stem there is no evidence of sclerosis at the junction of the medulla and pons, nor throughout the lower pons.

Pons; Upper Part of Middle Peduncles.—Two small oval areas of sclerosis are present in sections at this level—the one, lateral to the median raphé extends backwards and outwards from the surface of the pons and cuts across, in sharply defined lines, the grey matter, the transverse fibres, and a few of the fibres of the pyramidal bundles of the left side; the other area involves a small portion of the white matter of the cerebellum at the cerebello-pontine angle, together with the fibres of the medullary core and radiations of the adjoining folia. Both of these areas are absent at a slightly higher level, and the only lesion present is a small patch involving the intramedullary portion of the right fifth nerve immediately in its entrance. This was the only cranial nerve root which entered into or emerged from demyelinated tissue.

The outstanding points in the pathological condition are therefore (1) that the examination of the areas showed that the case was a typical one of disseminated sclerosis; (2) that in several

segments there was in the right lateral column, involving the crossed pyramidal tract, but not confined to it, an advanced sclerotic patch: (3) that in the left lateral column, in many segments, there was evidence of "shadow" sclerosis involving the crossed pyramidal tract; (4) that in other segments of the cord the lateral columns, including the crossed pyramidal tracts, were absolutely normal.

Variability of the early symptoms of the disease is one of the most striking characteristics of disseminated sclerosis. There is a tendency, however, for the patches to affect first the systems which are extensive, and thus the pyramidal fibres in their long course from the cerebral cortex to the anterior horn cells are often early involved. This involvement is usually progressive, and a spastic paraplegia develops. If the sensory fibres adjacent to the descending motor tracts are involved, well-defined sensory changes may result.

The absence in this case of symptoms, due to the involvement of the higher centres, the chief of which may largely be referred to want of co-ordination, may be explained by the absence, so far as can be ascertained, of sclerosis spreading in from the ventricles. The examination of the medulla and pons gave no evidence of periventricular lesions—a localisation which is the dominant feature in many cases.

THE ACTION OF VERATRONE IN THE TREATMENT OF ECLAMPSIA: A RECORD OF THREE CASES.

By W. F. THEODORE HAULTAIN, B.A., M.B., B.C. (Cantab.).

ECLAMPSIA is one of the commonest and gravest complications of pregnancy. Curiously, in some parts of Great Britain it is almost unknown, and should it occur in these districts the attack is usually slight and recovery the rule, whereas in other parts the disease is common, and the attacks are fraught with the greatest danger. It might be said that eclampsia is most common and most dangerous the further north and the further east one goes. Thus at the Royal Maternity Hospital, Edinburgh, eclampsia is a disease to be dreaded and feared. Into the above hospital are received most of the abnormal cases from the south-east of Scotland, and on looking at the statistics regarding the frequency of eclampsia one finds that annually there are admitted over 40 cases of eclampsia or threatened eclampsia, out of a total of about 700 indoor cases. Until recently the maternal death-rate was between

25 per cent. to 30 per cent., and the fœtal death-rate about 75 per cent. By the use of veratrone the death-rate in 38 consecutive cases was two, and the fœtal death-rate lower than 50 per cent. This shows a great improvement, which is further enhanced by the fact that neither of the two deaths was actually due to eclampsia, as one was from pneumonia and the other from puerperal septicæmia, which occurred after the actual eclampsia had been cured. So it might be said that veratrone had an unblemished record in these 38 cases.

Veratrone is a solution of the active principles of veratrium viridi, and is one-quarter the strength of the fluid extract, and one-twentieth stronger than the British Pharmacopœal tincture veratrii viridi of 1885. It is usually given hypodermically in doses of ½ to 1 c.c., but can also be given by the mouth if desired, in doses of 1 to 3 minims thrice daily. The latter method of administration is, however, very seldom used.

Many prodromal signs and symptoms are cited which warn the doctor or nurse that eclampsia is imminent. These consist of œdema of the extremities, puffiness under the eyes, albuminuria, scanty urine, flashes of light across the eyes, and severe headache. Some of these are always present in eclampsia, but in some cases all of them may be found, yet eclampsia never occurs. There are four cardinal symptoms, which taken together always denote that eclampsia is extremely near, but taken separately mean little. These symptoms are headache, high blood-pressure, very scanty urine, and albuminuria. As regards high blood-pressure, it is difficult to specify any fixed height and to say that above that height eclampsia will occur and below it will not; each case varies, as will be seen in the cases cited below. I might, however, state that 165 mm. is the minimum blood-pressure that I have noted when convulsions occurred. It is frequently considerably over 200 mm. The amount of urine per diem, when convulsions occur, rarely exceeds 10 ounces, and is usually less, whilst the quantity of albumen in the urine may be either very small or large, usually the latter.

High blood-pressure is perhaps the principal factor concerned, and if it can be lowered satisfactorily, convulsions will not occur; the urine will increase and the albumen present decrease. Veratrone acts, partly at least, by lowering the blood-pressure at an astonishingly rapid rate; it may fall 40 mm. to 60 mm. in less than thirty minutes. According to Dr. W. Cramer, who has been working lately on the pharmacological action of the drug—"the

fall of blood-pressure is due to a selective action on the afferent nerve endings of the vagus, which produces reflexly a slowing of respiration and a slight fall of blood-pressure, due to vaso-dilatation. As a rule there is also a marked slowing of the heart-beat, produced reflexly through normal inhibition." In a normal animal this is how the drug acts, but it would appear that in eclampsia it has some other action which is more specific.

It has been proved experimentally on healthy rabbits and cats that veratrone only slightly reduces the blood-pressure, and it was not until the animal had been injected with the toxine from an eclamptic and the blood-pressure thus artificially raised, that the drug had much effect in reducing it. The action might thus be compared with phenacetin and acetanilide, which only lower the febrile temperature and have no effect on the temperature in health.

If veratrone acted solely on the afferent vagal nerve endings it would produce its effect in health as well as disease, so probably it must have some other action which only occurs in cases of eclampsia. This is also shown by the fact that doses which are poisonous in cases of uræmia can be given with impunity in eclampsia, and also doses which are poisonous in cases of chronic Bright's disease with a high blood-pressure can be given safely in uræmia. Thus it would seem that the drug must have a specific action in both uræmia and eclampsia, but to a much larger extent in the latter.

Eclampsia is now said to be due to endotoxins, which are contained in the chorionic villi and syncytium, pass into the blood and give rise to an increase of the fibrogen and fibrin ferment content of the blood, caused by a decrease in the antithrombin, produced by the liver (Dienst). This would give rise to a coagulation or at least an increased viscosity of the blood, and which is borne out by the fact that at autopsies in cases of eclampsia the most striking feature is a periportal necrosis in the liver, due to thromboses, and to the agglutination of the red blood-corpuscles. Thromboses are also found in the brain. This also complies with the clinical features, as the blood-pressure always rises enormously and very little urine is passed, and these facts could be due alone to the increased liability of the blood to coagulate.

Veratrone lowers the blood-pressure, and this may be done in one of three ways, so far as its action on the toxins are concerned. It may prevent the formation of toxins by the chorionic villi and syncytium; it may act as an antitoxin in the blood, thus preventing

the toxins from reaching the liver, or it may act directly on the liver, stimulating it to produce antithrombin, and thus obviate the state of increasing coagulability of the blood. Which of these actions it has I am not prepared to state, but it undoubtedly has some very powerful specific action on the eclamptic state, as well as its more feeble action on the vagal afferent nerve endings.

It would appear to be a direct antidote to the poison of eclampsia, in so far as blood-pressure is concerned. At the same time the poison of eclampsia would appear to counteract the poisonous action of veratrone, and thus permit, indeed necessitate, it being given in much larger doses than can be given in the non-eclamptic state.

Three cases of eclampsia, which all have different important points, show how veratrone may be used in different cases:—

Case I.—Mrs. L., 24 years: primipara. Last menstrual period 14th to 21st May 1915. Admitted 4th January 1916.

History.—Had been attending a doctor since 24th December, on account of albuminuria. She has been put on milk diet and the usual medicinal treatment. The albumen, however, increased instead of decreasing until 2nd January, when she seemed to be getting better. On getting out of bed on 4th January the patient had a severe frontal headache and was obliged to return to bed. She also noticed that she passed an exceedingly small quantity of urine during that day. At 3.30 P.M. she took an eclamptic convulsion, which was very severe. At 4 P.M. another convulsion occurred. The doctor was called, and immediately ordered an ambulance to bring her to hospital. The journey took a little over an hour, and during that time she had three convulsions.

On Admission.—The patient was semi-conscious, her face was puffy, and her eyelids very oedematous. The foetal heart was still beating strongly and well. The pulse-rate was 136 and her blood-pressure 232 mm. On passing a catheter no urine was found in the bladder.

Treatment and Progress.—1 c.c. of veratrone was injected into her arm at 6 P.M. and at 6.15 her blood-pressure had dropped 20 mm. She, however, took another fit at 6.20 P.M., which was said to be not nearly so severe as the previous fits. At 6.30 P.M. her blood pressure had dropped to 132 mm. and her pulse-rate to 52, and at 7 P.M. her blood-pressure was 120 mm.

5th January.—Patient had no headache: her blood-pressure was again 200 mm., but her pulse-rate was only 72. One ounce of urine was obtained and was found to contain 5·4 grains of albumen to the ounce, and granular casts were present. Milk diet alone was given.

6th January.—At 1.45 P.M. the blood-pressure was 216 mm. and

pulse-rate 72, so ½ c.c. of veratrone was injected as a prophylactic, and the blood-pressure and pulse-rates were as under, at the times stated :—

2.15 P.M.	Blood-pressure 156 mm.	Pulse-rate 44
3 „	„ 140 „	„ 52
7 „	„ 172 „	„ 72

She only passed 5 ounces of urine, but her bowels had been moved five times by salts, and thus perhaps a quantity of urine was lost. It contained 5·3 grs. to the ounce of albumen.

7th January.—The patient felt almost well again. The swelling under her eyes had almost disappeared, and the œdema of her legs was much improved.

Blood-pressure and pulse-rate as follows :—

1.40 P.M.	Blood-pressure 210 mm.	Pulse-rate 60

She received ½ c.c. of veratrone.

2.10 P.M.	Blood-pressure 200 mm.	Pulse-rate 50
6.30 „	„ 182 „	„ 54

She passed over 6 ounces of urine, a large quantity being lost in the stools. Before the injection of veratrone the urine contained 6·2 grs. to the ounce of albumen, and after the injection 3·8 grs. per ounce.

8th January.—She received ½ c.c. of veratrone at 10 A.M.

10 A.M.	Blood-pressure 232 mm.	Pulse-rate 64
11 „	„ 130 „	„ 42
1 P.M.	„ 170 „	„ 46
8.30 „	„ 220 „	„ 50

She passed 27 ounces of urine, which contained not nearly so many casts as previously. Before the injection the urine contained 4·4 grs. to the ounce of albumen, and after the injection 2·4 grs. per ounce.

9th January.—She received ½ c.c. of veratrone at 10 A.M.

10 A.M.	Blood-pressure 208 mm.	Pulse-rate 64
10.45 A.M.	„ 88 „	„ 48
12 midday	„ 106 „	„ 34
1.15 P.M.	„ 122 „	„ 40
6.30 „	„ 180 „	„ 56

She passed 11 ounces of urine, which contained 1·8 gr. of albumen to the ounce of urine before the injection, and 1·5 gr. to the ounce after injection. All traces of œdema had disappeared and the fœtal heart was still heard, and movements were felt by the mother.

10th January. No veratrone was given on account of the collapse —the blood-pressure falling 120 mm.—which occurred after it the previous day. The blood-pressure was 208 mm. all day and the pulse-

rate 60. She passed 61 ounces of urine, which contained 5 grs. of albumen to the ounce in the morning and 1·8 in the afternoon.

11th January.—Again no veratrone was given. Blood-pressure 212 mm. Pulse-rate 72 all day. She passed 37 ounces of urine, the albumen content being 5·3 grs. per ounce.

12th January.—Veratrone 0·25 c.c., injected at 10.30 A.M.

10.30 A.M.	Blood-pressure 190 mm.	Pulse-rate 76
11.30 „	„ 140 „	„ 68
12.30 P.M.	„ 148 „	„ 68
7 „	„ 172 „	„ 68

She passed 41 ounces of urine with 1·4 grs. per ounce of albumen.

13th January.—Blood pressure 182 mm., so veratrone was not given. Also the foetal heart could not be heard. She passed 45 ounces of urine with an albumen content of 4 grs. per ounce.

14th January.—Blood pressure 176 mm. She passed 33 ounces of urine containing 3·1 grs. per ounce of albumen.

17th January.—Blood-pressure 166 mm. Urine contained 2·8 grs. per ounce of albumen.

18th January.—The patient gave birth to a dead foetus at 2 A.M. The blood-pressure after delivery was 160 mm.

From then onwards the blood-pressure fell and the albumen steadily diminished in the urine, till on 5th February the blood-pressure was 120 mm. and there was no albumen in the urine. She was discharged from the hospital feeling perfectly well.

In this case veratrone was first used as a cure for eclampsia, and it did its work well, as 1 c.c. alone was sufficient to control the fits. Later it was used as a preventative, and I think that the patient would most assuredly have had other attacks had it not been given almost daily. The child died, which was unfortunate, but if the attacks had been later in the pregnancy, which is more usual, the probability would have been that a live child would have been born. It is interesting to notice that after the death of the child on 13th January the blood-pressure began to fall slowly, and thus no further injection of veratrone was necessary. This is generally found to be the case, as the living foetus seems to supply the toxin which causes eclampsia. It is also noteworthy that before the death of the child when veratrone was given (11th January) the albumen content in the urine rose from 0·5 gr. per ounce to 5·3 gr. per ounce, although the amount of urine passed was only slightly less. Therefore to sum up this case, I would state that it was a fairly bad case of eclampsia, which was rapidly becoming worse. It was cured by veratrone, and the disease prevented from recurring by daily prophylactic

injections. Thus the mother's life was saved, but the baby died. This, however, was only to be expected, as the toxins circulating in the system for over ten days would be bound to have an ill effect upon it.

Case II.—Mrs. H., 34 years; primipara. Admitted 27th February 1916. Last menstrual period in May 1915.

History.—The patient had suffered since the beginning of February from swollen legs and a puffiness under the eyes. She paid no attention to these symptoms, as she felt quite well. On 25th February she began to feel a certain heaviness in the head, and noticed that she was passing very little urine. On 27th February, when she was eating her breakfast, she took an eclamptic seizure. She took another one an hour later and a third in the ambulance on the way to hospital.

On Admission.—The patient was quite unconscious, her legs were very œdematous, and there was a distinct puffiness under her eyes. She had a somewhat bloated appearance. Her blood-pressure was 168 mm. and the fœtal heart could be heard distinctly.

Treatment and Progress.—She was admitted at 2.15 P.M. and received 0·5 c.c. of veratrone subcutaneously. At 2.30 she took another convulsion and received another 0·5 c.c. of veratrone. Another convulsion occurred at 2.45 P.M., when she received another 0·5 c.c. of veratrone. Blood-pressure at 3 P.M. was 80 mm. and pulse-rate 48, and at 11.15 P.M. the blood-pressure had risen to 152 mm. and pulse-rate to 72. She had a catheter passed and 6 ounces of urine were drawn off containing 4 grs. of albumen to the ounce.

28th February.—The patient went into labour at 10 A.M. and was immediately put under the "new hyoscine-morphine" treatment. At 10.15 A.M. she received ¼ c.c. of veratrone. Her blood-pressure and pulse-rate were as under:—

10.15 A.M.	Blood-pressure	166 mm.	Pulse-rate	90
12 ,,	,,	104 ,,	,,	82
2 P.M.	,,	146 ,,	,,	92
3 ,,	,,	164 ,,	,,	118
4.45 ,,	,,	166 ,,	,,	118

She received 0·25 c.c. of veratrone at 4.45 P.M.

5.15 P.M.	Blood-pressure	128 mm.	Pulse-rate	84
8.15 ,,	,,	164 ,,	,,	90
9.45 ,,	,,	180 ,,	,,	120

She was delivered of a live child by forceps at 10.45 P.M.

11.40 P.M.	Blood-pressure	180 mm.	Pulse-rate 96

She received 0·25 c.c. of veratrone at 11.45 P.M.

12.45 P.M.	Blood-pressure	140.	Pulse-rate 84

She passed 11 ounces of urine during the day and the albumen was 1·1 gr. per ounce before the injection of veratrone and 0·7 gr. per ounce after the injection.

29th February.—She received 0·25 c.c. veratrone at 10.20 A.M.

10.20 A.M.	Blood-pressure 170 mm.	Pulse-rate 80
11.20 „	„ 124 „	„ 68
4.30 „	„ 170 „	„ 80

She passed 40 ounces of urine and the albumen contained was 0·15 gr. per ounce.

1st March.—The blood-pressure was still 170, but on account of the large quantity of urine that was being passed, and also the scantiness of albumen present, no further injections of veratrone were given.

The blood-pressure fell slowly, but did not reach its normal limit—120 mm.—until the 11th March. The albumen had all disappeared from her urine and she was feeling very well, and the baby was thriving and gaining in weight. She was discharged on 11th March.

This case shows how veratrone has to be used in some cases in successive doses before a cure may be effected, and if this had not been done the case would have been put down as one of the failures of veratrone. It was also used as a preventative while she was in labour; had it not been used, the patient would have been very liable to take convulsions. It is also an interesting case from the point of view that the blood-pressure which caused convulsions to occur was not very high, only being 166 mm., and I gave as my minimum 165 mm. The case is also noteworthy, as as it shows that the hyoscine-morphine and veratrone treatments may be combined with impunity, and it is interesting to note that the patient remembered nothing that occurred between breakfast time on 27th February till 6 A.M. on 29th February, and refused to believe that she had been at all seriously ill, and complained of being put on milk diet. The amnesia was no doubt entirely due to the scopolamine, as I have noted in many other cases.

CASE III.—Mrs. K., 25 years; multipara. Last menstrual period 20th May 1915. Admitted 11th February 1916.

History.—The patient was sent to hospital by her doctor because he had found that her urine had been loaded with albumen for a fortnight. She had been put on milk diet, but this did not ameliorate the symptoms, and she complained incessantly of headache, so he was naturally afraid of eclampsia occurring.

On Admission.—The patient was very puffy under the eyes and her legs were very œdematous. She also complained of headache.

Hydramnios was present, and the fœtal heart could just be heard. Her blood-pressure was 180 mm. and pulse 82.

Treatment and Progress.—She received ¼ c.c. of veratrone at 2.15 P.M., and at 3.15 P.M. her blood-pressure was 130 and pulse-rate 68, and at 9.30 P.M. the blood-pressure was 156 mm. The headache had almost disappeared in the evening. She only passed 9 ounces of urine, and it contained 12·2 grs. of albumen to the ounce.

12*th February.*—At 10 A.M. her blood-pressure was 170 mm., and pulse 80, so she received ¼ c.c. of veratrone, and at 11 A.M. the blood-pressure was 150 mm. She passed 18 ounces of urine and the albumen content was 7 grs. per ounce. Her headache was much better.

13*th February.*—At 10 A.M. her blood-pressure was 170 mm., and pulse 80, so she received ¼ c.c. of veratrone, and at 11 A.M. the blood-pressure was 150 mm. She passed 18 ounces of urine and the albumen content was 7 grs. per ounce. Her headache was away and the œdema was almost better.

14*th February.*—Blood-pressure 165 mm. She passed 45 ounces of urine which contained 7·6 grs. of albumen to the ounce. No veratrone was given.

15*th February.*—The blood-pressure rose to 190 mm., so ¼ c.c. of veratrone was given at 11 A.M. and at midday the blood-pressure had fallen to 108 mm., but by 6.30 P.M. it was up again to 160 mm. She passed 40 ounces of urine containing 9·6 grs. of albumen to the ounce.

16*th February.*—At 10.30 A.M. the blood-pressure was 120 mm., but at 8.30 P.M. it had risen to 200 mm. She received veratrone ¼ c.c. at 8.50 and she vomited several times after it, and at 9.30 her blood-pressure had only fallen to 180 mm. She passsed 41 ounces of urine containing 6·1 grs. per ounce of albumen.

17*th February.*—At 6.30 P.M. the blood-pressure was 180 mm., so she received ⅛ c.c. of veratrone, this small dose being given to try and keep away the sickness which the larger dose had caused. At 7.30 P.M. the blood pressure fell to 164 mm. She had no nausea. She passed 40 ounces of urine containing 6·1 grs. of albumen to the ounce.

18*th February to* 21*st February.*—Her blood-pressure continued to be 180 mm. during these days, but as she was passing such large quantities of urine it was thought inadvisable to give her more veratrone, especially as she had shown an idiosyncrasy to the drug.

22*nd February.*—At 4 A.M. the patient gave birth to twins. The labour was uneventful, but after it she complained of a severe headache, and she took an eclamptic seizure—convulsion—at 5 A.M. Her blood-pressure was then 185 mm. She received ¼ c.c. of veratrone, and in 15 minutes the blood pressure was 158 mm., and she complained of headache and sickness all day. She took a second convulsion at 4.10 P.M. when her blood-pressure was 180, and she received ¼ c.c. of

veratrone. This only brought the blood pressure down 10 mm. in half an hour, so ½ was added, and the blood pressure fell to 149 mm. by 5.15 P.M. She vomited repeatedly after that for about an hour, but in the evening at 7.30 P.M. she felt much better and her headache was relieved. She only passed 3 ounces of urine containing 9 grs. of albumen per ounce.

23rd February.—She took a fit at 12.30 A.M., when her blood pressure was 175 mm. She was given no veratrone, but she took another fit at 1 A.M., her blood pressure being still at 175 mm. She then received 0·5 c.c. of veratrone, and in half an hour the blood-pressure fell to 130 mm. She was again very sick, but this passed off in an hour or two, and she fell into a quiet sleep. At 10.30 A.M. her blood-pressure was 144 mm. and she said she felt much better. She took no more fits, and in the evening her blood-pressure fell to 130 mm. She passed 32 ounces of urine containing 6·1 grs. of albumen to the ounce.

24th February.—Blood-pressure 120 mm., and she felt quite well all day until the evening, when she started vomiting coffee-grounds matter.

25th February.—She was still vomiting, and washing out her stomach did no good.

26th February.—The patient became delirious and then unconscious, and died at 9.15 P.M.

At the post-mortem examination a septic peritonitis, due to streptococcal infection, was found.

This case was unfortunate in many ways. First she became infected, and what would have been a good result from veratrone became a death from puerperal septicæmia. However, it will be noted that the fits had been stopped and the eclampsia cured before the symptoms of peritonitis occurred.

Secondly, the patient showed a decided idiosyncrasy to the drug, and thus only small doses could be given and not so many as one would have liked. If she had been a normal patient she would have received preventive doses of veratrone during her labour and then probably puerperal eclampsia would not have occurred, but in her case it was not considered safe to do so, and she had just to take her chance without the help of veratrone. However, the case shows how veratrone can be used as a prophylactic, as it kept the blood-pressure down, and the urine increased in quantity and the albumen fell. It is interesting to note that when veratrone had not been given for two days, namely, 13th and 14th February, the blood-pressure rose to 190 mm. on the 15th February, whereas when veratrone had been given it did not rise so high. I have appended below a table which shows how

the albumen in the urine was affected by veratrone, the quantity present rising at a rapid rate when veratrone was not given, even though the amount of urine was increasing, and falling as soon as veratrone was given.

11/2/16.	Urine 9 ozs.	12·2 grs. of albumen per oz.			Veratrone given.
12/2/16.	„ 18 „	7·0 „	„	„	No veratrone given.
13/2/16.	„ 27 „	4·4 „	„	„	„ „
14/2/16.	„ 40 „	7·6 „	„	„	Veratrone given.
15/2/16.	„ 40 „	9·6 „	„	„	„ „
16/2/16.	„ 40 „	6·1 „	„	„	„ „

I have given the above cases in detail to show the necessity of carefully watching the blood-pressure during the treatment. In several cases it has been stated to be useless in the treatment of convulsions. In these, however, usually a small dose has been given haphazard, without repetition, and no careful account of the blood-pressure has been made.

In these three typical cases the four cardinal symptoms mentioned above were present together. Veratrone in each case caused the blood-pressure to fall considerably, and further, had an action in increasing the amount of urine passed and decreasing the amount of albumen. This seems at first to be coincident with the fall in blood-pressure, but later there seems to be some specific effect, as the blood-pressure may rise again but the urine never gets less, and the albumen always decreases after the drug has been given.

As regards the dosage of veratrone, I think that no fixed amount can be stated, as different people react quite differently to it. Thus in Case I. 1 c.c. was given with no bad effects whatever, whilst in Case III. ¼ c.c. caused vomiting and nausea. Case III. was, however, I think exceptional in her idiosyncrasy to the drug, and is the only case of the kind that I have heard of, but still it shows that one has to be on the alert for such cases, as a large dose might be exceedingly dangerous. I would state that though no fixed dose can be stated, the dosage appears to be as follows:—

Prophylactic dose, the patient never having had fits, but eclampsia imminent, 0·25 c.c. daily. Prophylactic dose, the patient having had fits and having been cured of these—0·25 c.c. to 0·5 c.c. daily. Dose when the patient has eclampsia and the fits are occurring—1 c.c. on admission and 1 c.c. after each fit. 0·5 c.c. might be given with benefit in the last case and an additional 0·5 c.c. given after each fit, but it is probable that although this will cure the disease in time the patient is liable to take many

more fits than she would have done if 1 c.c. had been used. This is well shown in Cases I. and II. Case I. had one injection of 1 c.c. and the fits stopped at once, whereas Case II. had to have three injections of 0·5 c.c. before the fits stopped, and thus she had two more fits than she would probably have had if 1 c.c. had been injected initially.

In the eclamptic state the drug seems to be able to be given with impunity in much larger doses than in any other condition. Thus in uræmia, for which I have used the drug also, 0·25 c.c. is quite sufficient to stop the fits, and 0·5 c.c. gives very untoward symptoms, such as weakening of pulse, sighing, respiration, and vomiting, which have to be immediately counteracted with strychnine.

In conclusion, it may be stated that we have in veratrone a drug of the utmost value in the treatment of eclampsia, as shown by its success in the treatment of 38 consecutive cases. After the initial dose of 1 c.c. subsequent doses should be regulated by the blood-pressure of the patient, as by so doing it can be given with safety and to the greatest advantage.

RECENT ADVANCES IN MEDICAL SCIENCE.

NEUROLOGY.

UNDER THE CHARGE OF

EDWIN BRAMWELL, M.B., F.R.C.P.

Myopathy and Traumatism.

Some four years ago the writer met with a case of myopathy in which the disease was attributed by the patient—a miner, aged 52—to a fall of coal which he stated had struck him on the back, between the shoulders. The muscles affected in the case in question at the time the man came under observation, some nine months after the alleged injury, were the serratus magnus, the latissimus dorsi, and pectoralis major. The condition was bilateral and more or less symmetrical. Both thighs were somewhat small in proportion to the calves. When the patient was asked to lie flat upon his back and then get up from the ground he turned over on his face and raised himself with his hands on his knees in the manner which is characteristic, but not pathognomonic, of certain types of muscular dystrophy. There was no facial weakness, no alteration in the reflexes, and no sensory loss. According to the patient's statement he had been perfectly well up to the time of the accident,

which occurred on the 6th May 1911, but from that date up to the 24th February 1912, when he was seen by the writer, he stated that he had suffered from pains in the back between the shoulders, and latterly from weakness in the arms, and had been consequently prevented from returning to work since the injury. At a later date the case became the subject of litigation, the patient claiming that his symptoms had been caused by the injury. Upon referring to the literature the writer was unable to find any evidence in support of the view that trauma may be the exciting factor in determining the onset of a myopathy; and, after carefully weighing all the available facts, the conclusion was come to that the apparent association was probably to be explained by coincidence.

Within the last few months the writer has, however, met with another case of myopathy affecting the muscles of the shoulder girdle, notably the trapezii, the latissimus dorsi, the sternal portions of the pectorals, and, to a lesser degree, the serratus magnus on both sides of the body, in which the patient—a soldier—stated that his symptoms had developed since he received an injury to his back. In this case the muscles of the lower limbs and all the muscles of the upper extremities, with the exception of those above mentioned, were unaffected. There was, however, a slight degree of bilateral facial weakness. According to the man's statement he had been buried some five months prior to the time when he was first seen by the writer, a portion of a trench and some sand-bags, which had been displaced by a shell, falling upon him and pinning him to the ground. Further, he stated that when he was passed for foreign service, eight months prior to the accident above referred to, he was examined, fully stripped, and passed without comment, while he also stated that up to the time of the injury he had felt perfectly fit, but that ever since that date he had complained of pains in the back and between the shoulders. The atrophy and consequent deformity of the shoulders were so pronounced when the case came under the writer's observation that it was obvious the man would never have been passed for active service had his symptoms been present to anything like the same degree when he was examined by the medical officer. The writer, on reviewing the facts at his disposal, felt justified in assuming, with a strong degree of probability, a relationship of cause and effect between the trauma and the myopathy. It is interesting to note the similarity in the distribution of the affected muscles in this case and in the case first referred to. These two observations afford at least very suggestive evidence that a myopathy of this type may be caused by a trauma, or at least that trauma may be the exciting factor in determining the incidence of the disease.

A paper by Claude, Vigoureux, and L'Hermitte (*Presse Médicale*, October 1915) affords corroborative evidence in support of this conclusion. These authors describe, under the title of "Muscular

Dystrophies of the Myopathic Type due to War Traumatisms," two cases of myopathy in which the distribution of the affected muscles was very similar to that in the cases above reported, the myopathy in these two cases being, in their opinion, directly provoked by a wound. The first case was that of a soldier who was wounded in the region of the shoulder. The point of entrance of the bullet or shrapnel was at the level of the upper part of the right acromio-clavicular articulation, while the missile had been extracted from the arm at the posterior border of the deltoid. There was in this case marked paralysis and wasting of the serratus magnus and trapezius muscles on the right side. It is unnecessary to consider here in detail the reasons advanced by the distinguished Parisian neurologists in support of their contention that the paralysis was of myopathic origin. The second case was that of a soldier who received a wound from a large piece of shell in the lower lumbar region. He suffered, in the first instance, from paralysis of all four limbs, apparently of functional origin, for he had completely recovered from these symptoms fifteen days later. When examined by the authors some seven months after the injury the man was found to present a very marked bilateral atrophy of the shoulder girdle, the serratus magnus and the trapezius being greatly atrophied, the latter more particularly in its lower two-thirds. The deltoid was also affected, especially on the right side, though to a less degree than the muscles previously mentioned. The upper limbs showed some diffuse wasting.

Both these cases were undoubted instances of myopathy: in both the symptoms, it was alleged, were first noticed after a trauma, nor was any evidence elicited which indicated that the myopathy might have been in existence previously; while, lastly, no other etiological factor, apart from the trauma, could be detected. The French authors remark that in the classical writings almost no allusion is made to a possible relationship between trauma and the myopathies. Although the problem, in view of the rarity of these cases, is perhaps a matter of scientific interest rather than of great practical moment, it is only right, in justice to the patient, that the significance of this relationship should be recognised. The authors make the suggestion that soldiers so affected should be discharged from the service, and that, in view of the fact that sufficient time has not elapsed to permit of a conclusion as to the subsequent course which these cases pursue, the soldier should be given a renewable gratuity, with an allowance for reduction of capacity for work of from 20 per cent. to 40 per cent. according to circumstances, while in two years' time he should be re-examined and the question of compensation reconsidered.

Another case of muscular dystrophy of the myopathic type following upon a war injury was demonstrated by M. J. Français at a recent meeting of the Neurological Society of Paris (*Presse Médicale*, 23rd

October 1916, p. 478). The patient in question presented atrophy of the face and shoulder girdle of a myopathic type, which had come on two months after a slight wound of the head. In the absence of a family history or other apparent cause the trauma appeared to be the only reasonable etiological factor.

The Treatment of the Causalgias by Injection of Alcohol into the Nerve Trunk.

The pain sometimes experienced as a consequence of a gun-shot wound of a peripheral nerve is most distressing and persistent. Weir Mitchell, so long ago as 1864, depicted in graphic language the agony endured by a soldier suffering from a wound of the median. No satisfactory explanation has as yet been advanced to account for the fact that wounds of the median and sciatic nerves are especially apt to be associated with severe pain, nor for the circumstance that in one case the patient's life is made almost unbearable because of pain, while in another case, in which the nerve presents an identical appearance, pain is not complained of. Fortunately the causalgias, as these painful nerve lesions are termed, are comparatively infrequent, for the pain which accompanies them may persist for many months, and the classical measures employed for the relief of this symptom, as all who have had experience of these cases know, are, with the exception of morphia, almost invariably useless. Recently injections of alcohol into the nerve trunk above the level of the wound have been employed, with most satisfactory results. It is found that an injection of 60 to 80 per cent. alcohol into the nerve above the level of the wound abolishes the pain. The peripheral nerves of the limbs, in association with wounds of which these causalgias occur, are mixed nerves. Consequently the procedure results not merely in sensory loss, but also in paralysis of those muscles which the particular nerve supplies. This is a fact which has to be borne in mind in considering the justifiability of injecting alcohol. Experience, however, shows that in many of the causalgias, in which the question of the advisability of alcoholic injection arises, there is already considerable paralysis of the corresponding muscles, while, further, the intensity of the pain in itself is often such as to render those muscles valueless which are not actually paralysed. Again, numerous observations have served to prove that the paralysis caused by an alcoholic injection passes off in the course of a few months, hence an injection of alcohol into a nerve trunk above the level of the wound is both justified and indicated in any case in which the pain is very intense and persistent.

Sicard (*Presse Médicale*, 1st June 1916) records his experience in this connection. Twenty-one cases have been so treated in his clinic with complete relief of pain. The individual nerves were as follows: —median, six cases; median and ulnar, three cases; sciatic, five cases;

brachial plexus, five cases; while in two instances the injection was carried out for the relief of pain associated with an amputation neuroma. Thirty-four cases similarly treated by other French observers are referred to.

The technique is simple. The nerve is exposed, preferably under general anæsthesia, and liberated from the surrounding cicatricial tissue. One or two centimetres of a 60 to 80 per cent. sterilised alcohol are injected by means of a fine needle into the nerve trunk three or four centimetres above the level of the lesion. An œdematous swelling is produced at the seat of the injection, the nerve trunk assuming a white appearance. With practice the whole transverse section may be injected by a single puncture; but, if this is found impossible, the nerve may be pricked in two or three places. E. B.

SURGERY.

UNDER THE CHARGE OF

J. W. STRUTHERS, F.R.C.S., AND D. P. D. WILKIE, F.R.C.S.

Rectal Wounds in the Present War.

Cuthbert Wallace (*Trans. Roy. Soc. of Med.*, 10th May 1916) divides wounds of the rectum into two classes—(*a*) those that complicate wounds of the buttocks, ischial fossæ, or perineum; and (*b*) those that are caused by missiles which traverse the pelvic portion of the abdomen. Both classes are often combined with fractures of the bony pelvis.

(*a*) The damage is likely to be much greater in the case of shell wounds, and a large portion of the gluteal region may be torn away. The sphincter and lower portion of the bowel may be completely torn away, or the lumen may be opened on one side only. In the case of small missiles, such as bullets, the sphincter may be intact and the lumen opened, or the tube perforated above that muscle. The peritoneum may or may not be opened. The chief danger is from septic absorption; but extensive wounds of the buttocks have done wonderfully well at the Front, as they are widely open and drainage is good.

The treatment of the lesser perforating wounds follows ordinary lines, and consists in getting free drainage by laying open the track or dividing the sphincter (Makins). If possible, an opening in the peritoneum is closed by sutures. The question of colostomy must be considered and performed if there is difficulty in keeping the patient clean and comfortable. It will be mostly required for cases in which the lower segment of the bowel has been carried away and the torn end lies patent in the pelvis. A transverse colostomy is recommended, as, if a permanent opening is required, the median transverse colostomy

is most convenient both for cleansing and for fitting a belt: further, if subsequent closure of the opening is probable, the freedom of movement of the transverse colon facilitates the operation.

(*b*) Wounds of the rectum proper and of the pelvic colon are liable to be complicated by wounds of the small gut and of the bladder.

The injuries to the small gut are likely to be severe and extensive, but if not too extensive, and if there has not been excessive haemorrhage, such lesions are less dangerous than are wounds of the rectum.

The damage to the rectum may be intra- or extra-peritoneal or both, and the missiles causing such an injury take a transverse, antero-posterior, or semivertical direction. It is important to remember that a missile, whose track from the position of the entrance and exit wounds seems likely to have missed the rectum posteriorly, not infrequently has injured the viscus.

When damage to the rectum proper or to the pelvic colon is diagnosed or suspected a laparotomy should be performed. The further procedure will depend on the extent of the wound in the bowel. If a small intraperitoneal wound is found it may be sutured. In the case of large wounds an iliac artificial anus has been found most satisfactory. The advisability of attempting repair of a large wound may often be settled by the general condition of the patient. In most cases a transverse colostomy is indicated unless it seems probable that the suture line will hold. If a retroperitoneal wound is found a transverse colostomy, with opening up of the wound, is the wisest course, and a drain down to the site of the lesion is advisable. Sepsis is more likely to form a local abscess in these cases than in lesions of the small intestine. A simultaneous colostomy and cystostomy, with suprapubic drainage, should be avoided if possible.

A fatal result is much to be feared from septic infection, even if the lesion is small and the condition of the patient good.

Fortunately rectal wounds are relatively uncommon. Out of a series of seventy three cases of wounds of the great gut there were only six cases of pelvic injury of the rectum proper, and only two of these could be sent to the base. Five patients died out of a series of nine cases of suture of the pelvic colon.

Sarcoma of the Intra-Abdominal Testis.

Grant (*Journ. Amer. Med. Assoc.*, 23rd September 1916) discusses the literature on the subject of sarcoma of the intra-abdominal testis, and records an interesting case of his own in which the growth was found irremovable at an exploratory laparotomy; but later, after a thorough course of treatment by Coley's fluid, the tumour became reduced in size and was successfully removed at a second operation.

Bulkley has collected from the literature fifty-seven cases of sarcoma of the intra-abdominal testis; thirty-seven of these cases

were single cryptorchids. Seventy five per cent. of all the cases occurred in patients between twenty five and forty five years of age, and 74 per cent. of the mortality occurred within one year of operation. The fatal progress was more rapid in the undescended than in the scrotal testis. Hinman states that malignancy is relatively more common in the undescended testis, and this view is generally accepted. According to Bulkley one in every four cases of malignant disease of an abnormally placed testis is in the abdomen, and there is one abdominal sarcoma to every fifteen sarcomas in the scrotum.

The majority of testicular neoplasms, according to Ewing, originate in the sex-cells and are teratomata. The teratomata and mixed types of tumours grow more slowly, and in these cases the lymph glands are not involved so early. When the disease is in the right testicle the glands of the vena cava are first involved, and when the left testicle is affected metastases occur primarily in the glands along the aorta.

Of the forty-seven cases of intra abdominal sarcoma operated upon only two patients were known to be alive after two years.

Syphilis, tuberculosis, and simple tumours of the abdominal testis are practically unknown.

The author's case was a patient of thirty-seven years with a large abdominal tumour on the right side, extending above the umbilicus and having a fixed point in the pelvis, with little lateral movement. A laparotomy was performed through a middle-line incision, but as the growth in the testis was so extensive, vascular, and fixed it was decided not to attempt removal. On the third day after the operation administration of the erysipelas and bacillus prodigiosus toxine was commenced (Coley's fluid). The initial dose was half a minim, and this was gradually increased until a reaction of 102° to 103·5° followed, an injection being given thrice weekly. During the first month the growth was stationary, but at the end of seven weeks there was a distinct decrease in its size.

During eleven months more than one hundred injections were given, the maximum dose being eighteen minims. The patient was also given an occasional exposure to X-rays.

During a period of three weeks the injection by toxines was suspended, and it was noted that the cessation of the treatment coincided with an increase of the size of the tumour, which, however, again diminished when the injections were resumed. This observation was subsequently repeated.

According to Coley it is advisable to inject the toxines directly into a sarcomatous tumour. This was done latterly in this case, but it was found that half the usual dose given subcutaneously was all that could be tolerated, as the febrile reaction was much severer.

After thirteen months' treatment the tumour was smaller, more circumscribed, and harder. A second operation was performed and the

tumour successfully removed. The growth was found to be quite uniform and regular in outline, the projecting nodular appearance noted at the first operation having disappeared. Numerous strong adhesions, however, had in the meantime developed, fixing the tumour to the peritoneum, omentum, and colon.

Apart from delay in healing, due to the large cavity left by removal of the growth, the patient's progress was excellent after the operation. Microscopically the tumour proved to be a typical large round-celled sarcoma with areas suggesting the appearance of a carcinoma; the growth, therefore, being of the mixed type and, probably, a malignant teratoma.

The cure of an advanced case of sarcoma of the testis, such as the one recorded, is exceptional and requires several years to determine. The author believes that the mixed toxine (Coley's fluid) in the case reported had a repressive or inhibitory effect on the tumour, and must have had an influence in producing the disappearance of the nodular character of the growth.

Unilateral Symptomless Hæmaturia.

Payne and MacNider (*Journ. Amer. Med. Assoc.*, 23rd September 1916) believe that the term "essential" applied to cases of symptomless hæmaturia should be discarded. In three human cases, and in six experimental cases, definite lesions in the medulla have been demonstrated. The actual source of the bleeding in these cases seems to be from dilated veins in the pyramids and on the free surface of the papillæ. In all three human cases the same pathological changes were present. Few, if any, changes were found in the cortex, but a marked overgrowth of chronic inflammatory tissue occurred in the medulla and pyramids around the vessels of this zone, and these vessels in turn were dilated and resembled varicosities. Experimentally, in the dog a condition of hæmorrhage analogous to the clinical type was established, and the changes found in the kidneys were similar.

According to the authors the sequence of events in the pathology of the condition giving rise to the so-called essential hæmaturia is as follows:—

1. An infection of the kidney takes place, the foci, however, being limited to the medulla: no areas of infection have occurred above the cortico-medullary junction.

2. The repair of the infected areas is followed by an overgrowth of dense fibrous tissue.

3. This fibrosis produces an obstruction to the venous return from the medulla and papillæ through the venulæ rectæ.

4. As a result of this obstruction varicosities develop, which not only appear in the substance of the papillæ and beneath the surface

epithelium, but may rupture on the free surface of the papillæ and thus give rise to hæmaturia.

Various procedures have been suggested for the treatment of this condition, such as nephropexy, decapsulation, nephrotomy, and nephrectomy.

Nephrotomy is the procedure which has yielded the best results. Nephrectomy should be restricted to cases in which nephrotomy has failed, or those rare cases in which the bleeding has produced a critical condition.

The authors have performed nephrotomy with bisection from pole to pole and down to the pelvis in eight cases, successfully. Careful suture of the kidney is necessary, otherwise there may be troublesome bleeding within forty-eight hours. J. M. G.

OBSTETRICS AND GYNECOLOGY.

UNDER THE CHARGE OF

A. H. F. BARBOUR, M.D., AND J. W. BALLANTYNE, M.D.

The Etiology of Puerperal Peritonitis.

Dr. H. Chassot of Geneva furnishes the profession with some valuable facts respecting the part played by the Fallopian tubes in the causation of puerperal peritonitis (*Corresp.-Blatt. für Schweizer Aerzte*, 19th August 1916, vol. xlvi. No. 34, pp. 1057-1069). There has always been some doubt as to the relationship between septic endometritis and septic peritonitis in cases of puerperal infection, and Dr. Chassot quotes a number of standard authors to illustrate the diversity of views prevailing at the present time. Whilst some obstetric writers regard puerperal endometritis as a common cause of salpingitis (early or late), and consider that the tubes are infected by the continuity of the mucous surfaces, and that through them the peritoneum is invaded; others lay less stress on tubal infection and inflammation, and see the uterine vessels and lymphatics and the parametrium as the parts most commonly involved in the transmission of the infection to the peritoneum. In order to reach some sure ground on which to base the treatment of puerperal septic peritonitis, Dr. Chassot has examined with scrupulous care the organs, and especially the Fallopian tubes, in nine cases of puerperal fever ending in death. In each instance a full clinical history is added. The results have been somewhat surprising, and they go far to prove that far too much emphasis has been laid on diseased tubes as the connecting link between septic endometritis and peritonitis in infected cases following abortions and labours. In only one of the cases (the ninth) was there pyosalpinx on one side, and in that instance the writer regarded it as secondary to an ulcer of old standing

on the uterine mucous membrane and not as the cause of the pyæmia which led to the death of the woman. In one other case only (the sixth) was there evidence of old-standing catarrh of the tube which could possibly have been the cause of the peritoneal infection. In three cases (the first, third, and fifth) the tubes were normal, showing not even catarrh, far less purulent inflammation. In all the others (the second, fourth, sixth, seventh, and eighth) there was indeed some catarrh, but it was slight or only to be detected by microscopical examination. The author does not consider that he is making an exaggerated statement when he affirms that, in nearly all the fatal cases of puerperal fever with peritonitis which he studied, there was no trace of macroscopic pyosalpinx. These facts militate against the idea that the tubes play an important part in the propagation of the puerperal infection from the uterus to the peritoneum; one must therefore think rather of the veins or of the lymphatics of the uterine wall and of the parametrium as the channels of infection. Dr. Chassot is rather at a loss to explain the absence of tubal infection, or its slight nature in puerperal endometritis: but he suggests some factors which may play a part. One is the small size of the tubal orifice at its uterine end; indeed, the orifice may be completely closed by the swelling of the inflamed endometrium. Another is the defensive action of the cilia of the tubal epithelium, which wave towards the uterine interior, and so tend to check the upward progress of the infecting agents. A third is the biological nature of pyogenic microbes, and especially of streptococci. They are rather the parasites of the tissues than of the secretions, and they grow better in the substance of the tissues than on the surface. Surface propagation is almost peculiar to the gonococci. But it may be asked, how can the slight tubal catarrh which the microscope discovered in some of the cases be accounted for? Dr. Chassot replies that the solution of the problem is quite easy. The tube lies bathed in the infected peritoneal secretion, and so the inflammation is propagated more or less deeply into its wall, and sets up the catarrh. On the whole, these carefully made observations go to show that post-abortum or post-partum septic endometritis is not often transmitted to the peritoneum by the continuity of the mucous membrane of the Fallopian tubes. Possibly, when there is evidence of such transmission by contiguity of structure, the infecting agent has been the gonococcus and not the streptococcus.

Uterine Perforation in Induced Abortion.

Dr. V. B. Beckman of Petrograd has reported two somewhat similar cases of accidental perforation of the uterus during the induction of abortion (*Ann. de gynéc. et d'obstét.*, 1916, vol. xi. 2nd ser. pp. 206-212). In one of these cases the history given was that a midwife had introduced a laminaria tent into the uterus of a woman who had

missed two periods; in the other the woman herself tried to procure abortion by passing a bougie into what she wrongly thought to be her pregnant uterus, for it turned out afterwards that conception had not occurred. When the former patient came into hospital suffering from the symptoms of peritonitis, a vaginal examination with the speculum revealed a silk thread protruding from the cervix uteri. This thread came away when pulled upon. The sound, passed into the uterus, detected no foreign body; but since there was much haemorrhage Dr. Beckman dilated the cervix and curetted the interior without discovering any perforation. The patient's general condition was good, but a finger passed into the womb discovered an irregularity of the anterior wall. A few hours later grave symptoms appeared, and it was decided to open the abdomen. When this was done bad-smelling pus exuded, and the laminaria tent was found lying above the symphysis pubis. The pouch of Douglas contained pus mixed with blood. After the fluid had been washed out the uterus and its annexa were removed; the vaginal mucosa was sutured to the peritoneum, the stumps of the broad ligaments were fixed to the lateral parts of the vagina, and a drain was led from Douglas' pouch into the vagina. Fever and the formation of an abscess followed the operation, but in the end the patient made a complete recovery. The second patient, like the first, was a multipara; she thought herself pregnant, and introduced a bougie, which she had found in the dispensary where she was employed, into her uterus. During its introduction the bougie slipped from her fingers and disappeared. Soon thereafter she began to suffer from pain, and four days later she applied for admission to the hospital. Dr. Beckman found the uterus anteflexed and not enlarged; the os was closed; but through the anterior cul-de-sac a foreign body resembling a bougie could be felt. She was put under chloroform and ether, the abdomen was opened, and the bougie was discovered lying under an adherent piece of thickened omentum. The uterus, which was smaller than normal, was removed, but the annexa were left. The uterus showed no trace of pregnancy, but there was a perforation the size of a pea on the right side in the fundus. As in the other case, the lower part of the abdominal wound was left open with a drain in it, and the peritoneum also was drained into the vagina. Some pus came away from the two openings for a few days, and then recovery followed. This case was, therefore, one of those known as the attempted induction of abortion in the absence of pregnancy (*Conatus abortus provocandi defuncte gravidilate*).

Cases such as these raise several questions of interest and importance, and to some of them Dr. Beckman draws attention. The symptomatology, for instance, is not characteristic, and it is not surprising that the discovery of foreign bodies in the peritoneal cavity has been often quite accidental. The diagnosis, too, is difficult, for in

addition to the indefiniteness of the symptoms there is the circumstance that sometimes the woman does not know what has been done to her. When, however, the diagnosis has been made, immediate operation is demanded. The best incision is doubtless that through the abdomen rather than vaginal section, for the former allows not only the easy removal of the foreign body, but also the examination of the intestine for the detection of perforations either in existence or merely threatening. It is impossible to fix general rules as to the removal of the uterus. If, however, both the uterus and the annexa are infected a total hysterectomy is indicated, for the woman's life is in danger. When there are no signs of infection it should be sufficient to remove the foreign body and suture the perforation. Dr. Beckman cites in support of this procedure Vasten's remarkable case in which he removed a bougie from the abdomen of a woman who was three months pregnant, and whose uterus showed a cicatrix on its posterior wall, and who nevertheless went to the full term of her gestation. Abdominal section is also indicated in the cases in which no foreign body can be felt, as likewise in those in which it lies high up; but when it is encysted in the pouch of Douglas a posterior colpotomy should be done, the uterus being left, save when it is infected, and then it can be taken away *per vaginam*. When the foreign body is in the peritoneal cavity and the abortion is still incomplete, the excessive bleeding may necessitate the rapid manual or instrumental emptying of the uterus. In that case one ought to proceed with care, otherwise one may push the finger or the curette through the opening, and even when no perforation can be felt, but is suspected, it will be well to avoid the use of an intra-uterine douche. Some of the recorded cases prove that delay is not necessarily fatal; yet the danger undoubtedly increases with the lapse of time. Probably it is wiser to intervene immediately.

Psychic Vaginismus.

Dr. P. H. Williams (*Amer. Journ. Obstet.*, August 1916, vol. lxxiv. pp. 226-232) would divide all cases of vaginismus into two groups — organic and psychic. Under the head of "organic" he includes all those which depend upon some ascertainable cause, such as a tender urethral carunele, irritable hymen, or ulcer and fissure in the vulva or lower part of the vagina. In the psychic group are placed those in which there is no ascertainable pathological lesion about the external genitals. At the same time it has to be borne in mind that from repeated attempts at coitus a condition of extreme irritability about the vaginal orifice may be set up in a case belonging to the psychic type; so, too, an actual neurosis may arise from a very small lesion in the mucosa in the organic form in a highly neurotic individual. Dr. Williams records two cases in which the vaginismus was due

apparently to mental and not to local causes. In one the inhibiting impulse was conscious, rather than subconscious, at first, and had been imparted by suggestion from a second person: in the other the inhibiting influence was the long continued suppression of a natural impulse by the exercise of an immoderate amount of will power, and later, after the voluntary suppression was removed, subconscious inhibition continued. In these psychic cases the symptoms are not helped by surgical means; they should be treated as neuroses or phobias. This does not mean their transference to the psychiatrist, Dr. Williams thinking that the gynecologist should be able to manage them himself. The diagnosis is the really difficult matter, and can only be arrived at by exclusion and the exercise of great patience. Sometimes the suggestion of an operation works a cure, and probably some so-called surgical cures have really been psychic effects: but they ought, as a general rule, to be regarded and treated as neuroses.

J. W. B.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

W. G. SYM, F.R.C.S., AND A. MACGILLIVRAY, M.D., D.Sc.

COLOURED AUDITION.

IT is a relief to turn for a short time to a subject entirely unconnected with war.

Coloured audition is a phenomenon whose authenticity can no longer be doubted, and though the ordinary level-headed individual may regard those who exhibit the symptom as, at the least, bordering upon the pathological and neurotic, yet sometimes at least these cannot be shown to belong to any definitely abnormal class. The subject was recently dealt with under the head of "L'Encéphalopsie chromatique," by M. Blanchard, at the Paris Académie de Médecine. He turns aside at the very beginning of the paper which he read to recall the curious concerts which used to be given in London, at which the colour-organ was played. This was an instrument which was played upon as an ordinary organ is, but the keys, instead of being connected to pipes, threw coloured rays of light upon a screen: instead of a feast of sound the "audience" was regaled with an orgy of colour. For long, in certain of the works dealing with orchestration, colour-phraseology has been employed: thus the trumpet is regarded as red and the clarinette blue. Perhaps, as Blanchard says, we shall come to regard certain musical compositions as possessing colour, and speak of Beethoven's Pastoral Symphony, for example, as "green."

M. Blanchard takes the opportunity to describe somewhat fully two cases with which he had recently met, which bear upon coloured

audition. One was that of a young lady whom he met casually, who associated certain numbers with the idea of colours. This appeared to be true of certain numbers only, and not to be invariably present. She had always done this—a fact which her relatives corroborated on inquiry—and he was unable to shake her consistency of statement.

Number	0	she	associated	with a white colour.
,,	1	,,	,,	a black colour.
,,	2	,,	,,	a pleasant bright red.
,,	3	,,	,,	grey.
,,	4	,,	,,	yellow.
,,	5	,,	,,	a bright grey.
,,	6	,,	,,	a pinkish grey.
,,	7	,,	,,	green.
,,	8	,,	,,	mauve.
,,	9	,,	,,	a grey brown.

Four years later, on inquiry, she gave practically the same colour-values to numbers, adding that 0, 1, 3, and 9 always conveyed also the idea of sadness, while the others suggested ideas of brightness and joyousness. The mixture of numbers was less defined perhaps in its reaction, but, on the whole, the conjunction of two "sad" numbers was also sad, of two "happy" numbers was also happy, and of one sad with one happy was neutral. Many composite numbers were not associated with any definite colour-ideas. In the case of this lady the symptom, present and consistent during childhood, adolescence, and youth, as ascertained partly by the accounts given by the family, and partly by investigation on three occasions at intervals of thirty-six and of more than twelve months, gradually faded out, a change which the writer attributed to marriage and all that that involves.

His second case also occurred in a young lady, an artist in colour. Her story was that from early childhood the name of the day of the week, whether read, spoken, or merely thought of, always awoke in her mind the idea of colour, and always the same for the same day. Thus Monday was green, Tuesday grey, Wednesday white, Thursday blue, Friday chestnut, Saturday black, and Sunday rose. Violet, indigo, yellow, orange, and red did not occur in her weekly scheme. The names of the months and the hours of the day produced no such reaction. This association of days with colours has continued since she was six years of age, and is in no way affected by the weather or atmospheric conditions. Thursday is blue and Saturday black, for example, whether the weather is bright or dull, rainy or frosty. She does not *see* the colour; objects are not tinted with it; it is purely psychical, but it never changes; and the phenomenon works the other way also—for example, green, whether emerald or sap, evokes the idea of Monday. He was able to satisfy himself that in all this there was neither fraud nor conscious simulation.

Cases such as these have close relation to the more familiar examples of coloured audition, that curious condition in which a normal stimulation of the auditory apparatus is interpreted by the individual both as a sound and as a sensation of colour—sound perception has a colour stimulus added to it. These luminous perceptions are called photisms, and the association, synæsthesia or synopsia. Sollier has shown that these are sometimes accompanied by sensation of taste, but this is much more rare. From these cases, however, the two related above differ in respect of their point of departure.

Francis Galton was the first, or one of the first, to draw attention to this matter in general—to these psychochromes, as he named those sensations of colour aroused by stimuli other than external visual in this condition of cerebral coloured vision (or *encéphalopsie chromatique*)—and the subject has from time to time attracted the attention of psychologists and other scientists. Some appear to have expressed astonishment that the phenomenon is not present among either the congenitally blind or the congenitally deaf, but the greater wonder would be if it were! Of course the author has been unable to escape Locke's famous blind man, who considered that scarlet must be like the sound of a trumpet. It would be interesting to know whether the man who expressed himself thus had been really congenitally blind; probably he was. He could only express his sensations in terms of those sensations which he was capable of appreciating, not in those of which he was entirely ignorant, and into his phraseology must not be read more than it can carry.

A curious point about those who associate colours with numbers is that there appears to be no rule applicable to a number of cases regulating the association. One will associate red with 2, another red with 5, another green with 5.

Some associate the letters of the alphabet with colours, more especially the vowels, and here, again, there is no real regularity, for even if, as stated by some, the letter *i* is usually red to these people, others have noted cases in which *i* was blue, was bright blue, was yellow, had the colour of mercury, was white. The little poem by Arthur Rimbaud is interesting in this connection.

A noir, E blanc, I rouge, U vert, O bleu, voyelles,
Je dirai quelque jour vos naissances latentes.
A, noir corset velu des Mouches éclatantes
Qui bombillent autour des puanteurs cruelles,

Golfes d'ombre ; E, candeur des vapeurs et des tentes,
Lance des glaciers fiers, rois blancs, frissons d'ombelles :
I, pourpres, sang craché, rire des lèvres belles
Dans la colère ou les ivresses pénitentes :

U, cycles, vibrements divins des mers virides,
Paix des pâtis semés d'animaux, paix des rides
Que l'alchimie imprime aux grands fronts studieux ;

O, suprême Clairon, plein de strideurs étranges,
Silences traversés des mondes et des anges :
—O l'Oméga, rayon violet de Ses Yeux !

Another aspect of this "colouration" of non-materials is given in an expression of Khikmet Bey, a well-known literary Turk, who writes: "I have always thought each religion has its peculiar colour —the Jewish, black; the Christian, blue; the Mohammedan, red." Again, in 1759 a monthly magazine, called *Le Journal à la mode*, appeared with the announcement that each (monthly) issue would appear in the colour appropriate to that month—January in black, February in brown, and so on, with the remaining months in grey, green, lilac, poppy colour, crimson, blue, violet, yellow, golden, russet.

Colour enters more than a little into our phraseology regarding the various mental states, and such expressions are sanctioned by the agreement of the many in their employment; for example, one may "see red," be in a "blue funk," take a rosy view of the situation, or entertain black views. These phrases have taken their origin, says Blanchard, in *encéphalopsie chromatique*.

The author concludes that while it is in virtue of the association intracerebrally of the visual and auditory apparatus that *encéphalopsie chromatique* takes place, yet to attribute the phenomena to the simple anatomical fact (as some have done) is not justifiable—the matter is too complex for that (*Bulletin de l'Académie de Médecine*, Paris, May 1916).

[Perhaps the reviewer should apologise for the introduction into this account of the progress of ophthalmology the discussion of a matter which belongs rather to the domain of psychology.]

W. G. S.

DERMATOLOGY.

UNDER THE CHARGE OF

R. CRANSTON LOW, M.B., F.R.C.P., AND F. GARDINER, M.D., F.R.C.S.

Anaphylactic Food Reactions in Skin Diseases.

Dr. Albert Strickler (*New York Med. Journ.*, 29th July 1916) in an article on this subject attempts to put on a scientific basis what most dermatologists have been trying to do clinically. More and more it is borne on one that certain foods have a pathological action in producing or aggravating certain dermatoses, and this author's method, if found by further experience to be reliable, should prove valuable.

Briefly described, his plan is to extract the proteins from certain foods by weak alkali, incubate, filter, treat with absolute alcohol, evaporate, and then make a saturated solution in alkalinised saline. The solution thus obtained is tested for sterility and its nitrogen content ascertained. By experiment he has found that $\frac{1}{10}$ c.c. was locally effective when introduced endermically, and gave rise to no constitutional symptoms. As some reaction always follows, the following standards were adopted :—

1. The lesions must persist for 24 hours.
2. A distinct papule must be formed.
3. Erythema must be present.
4. Presence of tenderness at the point of injection.

What appears to be a quicker, but evidently, from the tone of the article, a less reliable method is to introduce the protein by a dental burr, employing a control at the same time. The local reaction thus obtained reaches its height in 30 minutes in the form of a papule—never a wheal.

Another competent observer, viz. White of Boston, has studied the reaction in 56 cases of eczema, and his conclusions are :—

(*a*) Eczema in children is due in a percentage of cases to excessive fat or sugar in their diet, or to egg albumin or milk, while in a minority of instances there is diminished thyroid secretion. Twenty per cent. of eczema cases are not influenced by food.

(*b*) Normal people do not react to these tests.

(*c*) Food anaphylaxis may be a factor in urticaria and, possibly also, in acne vulgaris.

(*d*) In eczematous individuals tested, about 30 per cent. gave positive results.

Strickler goes further afield, applying the reaction to other skin conditions, and also gives results of treatment as indicated by these tests :—

1. Forty six cases of eczema experimented on showed 12 who gave no positive reaction : and out of the 34 where reaction gave a cue to treatment, 23 were more or less benefited by the alteration in diet, the remaining 11 deriving no benefit.

2. Ten cases of urticaria of which 8 gave a positive reaction : and it is of interest that only 3 were acute, and that they were the only cases positive and benefiting by change of diet.

3. Eleven cases of psoriasis with 4 positive reactions, but in only one of these did improvement follow on the corrected diet.

4. Thirteen cases of acne vulgaris, 3 of acne rosacea with 11 positive reactions, but the change of diet, although persisted in, was without result.

The author concludes that the test is of value for diagnosis and treatment, and that the greatest benefit is derived in eczema but

disappointing in other dermatoses. He wisely adds that further experience is necessary.

The point of criticism that naturally arises is whether these local reactions are or are not necessarily indications that the food is the causative factor, and are there not other factors of more or less importance in individual cases? Apropos of these other factors Hermann (*Journ. of Cut. Dis.*, April 1916) discusses *in extenso* the theories about eczema. That new dermatitis is but old eczema writ large has long been our opinion, and he comes laboriously to the same conclusion, but he appears not to realise that the former is also more in line with modern scientific terminology.

He very clearly emphasises the modern view, which, while not ignoring the external irritant, does not neglect the effects of general systemic disturbance. The consideration of the relationship of dermatitis to other dermatoses is less convincingly discussed. The British School is blamed for placing seborrhœic dermatitis in its nomenclature instead of seborrhœic eczema; but then, as indicated above, he considers them the same; the truth evidently being that he loves to keep the dear old word eczema. It is rather late in the day to be separating prurigo and lichen from the category of dermatitis or eczema, and if he admits that eczema frequently arises in seborrhœal areas it appears to us still better and more accurate to call these eruptions inflamed seborrhœas—or better, seborrhœic dermatitis. Some of the cases he describes—and he takes 14 as typical examples—come obviously into this category, and others are purely dermatitis due to external causes.

Both internal and external causes are, in his opinion, responsible, and he boldly remarks "the important fact is that neither without the other will suffice"; and it is the practical problem of the student of eczema to try and determine the nature of its causes, for without doing so he cannot treat the disease rationally.

This generalisation is fairly accurate in many extensive and chronic cases, and, in these, general systemic treatment, whether by diet or medicine, the physician would be foolish to neglect, but, on the other hand, as assuredly we come across numerous examples where there are only local irritants producing the eruption.

Occupations as Contributing Factors to Diseases of the Skin.

This is the subject of a lengthy article by C. J. White (*Boston Med. and Surg. Journ.*, 13th July 1916).

Diseases of the tropics crop up here as in America, and although we may not see them very frequently, yet every now and then we come across, and should be on the look out for, Oriental sore, elephantiasis, leprosy, Dobies' itch, etc., and it may be that after the war there will be more of these cases amongst soldiers returning to civil life.

Cold as a factor is more noteworthy here than heat, but light prurigo and the extreme form of light and sensitiveness seen in xeroderma pigmentosum are well known.

The term "sailor's skin" is not much in use here, and yet, as he says, "You have all noticed the peculiar skin of hands, forearms, and especially the neck of farmers, gardeners, trench diggers, fishermen, and sailors. You have observed the brown colour, the thickness, and, in the case of the neck, the deep creases and at times tortuous folds of the skin. These conditions are common, but in certain individuals the sun produces more important changes. The skin of the face or the back of the hands may become in places a little darker brown, and rough and scaly—a condition which we call keratosis: and as a further development, cancer may arise *de novo*, or out of these localised keratoses."

We agree that light has probably to do with their production, but as these keratoses are one of the initial stages of rodents, they are more typical of skin senility. Like all rodent ulcers, they are more common on the face, but we have seen them on places like the back and chest protected from light.

A case of an old "salt" with 10 of these rodents on his face and neck came under our observation, happily yielding readily to treatment by CO^2 and X-rays.

Among diseases of posture, varicose dermatitis, erythema induratum (Bazin's disease), and the callouses of tailors and shoemakers are well known.

Insects, vegetable parasites, and bacteria are often incidentally infective agents in certain occupations, and we would almost class scabies and pediculosis as an occupational eruption of military life at present; the more serious diseases, however, are actinomycosis, sporotrichosis, and blastomycosis, got from grain or cattle.

Foot-and-mouth disease is recognised as affecting human beings, and evidently the author attributes butcher's pemphigus to this cause in a graphic description:—"Here and there in affected districts men who have dealt with diseased animals have developed a peculiar malady in which their bodies and the insides of their mouths are covered with great water blisters which come and come, and sooner or later the man dies mysteriously."

Chemical irritants take a large place in this instructive paper, and those who are interested may well consult his list. Forty-six diseases among 120 different occupations are mentioned and briefly discussed, and he, from his own statistics and those of others, claims that $\frac{1}{4}$ to $\frac{1}{3}$ of all cases of eczema are directly due to the occupation of the individual. The estimate, in our opinion, is much below the Scottish standard. The evil is a great one, and can be prevented or mitigated "mainly through the education of the employer and employed. The

average employer is not a brute, and in any case it is not to his interest to have his men incapacitated." "One great object in the present extensive study of occupational dermatoses is to teach the employer in every possible way how to safeguard the health of those in his employ, and this purpose is slowly but surely being evolved." He does not mention the matter, but it is a fact, in this country at least, that while heart, lungs, and general physique are considered by the medical examiner before a worker is admitted to a factory, the condition of the skin is ignored. It is as absurd a state of matters as if we ignored the state of and the manning of the first line of trenches in war.

Many men and women are seen at out-patient departments of hospitals who have skins of such a type or so diseased that they are entirely unfit to stand the irritation of their work, and should never have been allowed to commence their training.

Poisoning by Crude Coal Tar.

It is of importance that this should be recognised as of possible occurrence. Milder degrees we have from time to time observed in which headache, sickness, and some slight feverishness has occurred, but he mentions severe cases.

In the experience of C. J. White (*Amer. Journ. of Cut. Dis.*, July 1916) full strength tar is over-stimulating, and he uses the following:—

℞ Crude coal tar.
Zinc oxide āā 2.
Amyl.
Vaseline āā 16.

This he applies "twice daily under a layer of thin compress until the moist lesion has dried up and disappeared, or until, as happens in a certain proportion of the patients, papules begin to develop *in situ*," as these are apt to become pustular at a later stage.

He states that he has used this in every moist non-pustular condition of the skin, and up to a few weeks before writing had no trouble. Then the following cases occurred:—

1. A man with moist eczema of face, trunk, and extremities was treated for two days with tar in proportion of 4:32, but then, as the skin became irritated, 1:30:32 was used. On the fifth day after admission he complained of uneasiness, and next day exhibited a temperature of 101·5 and a general itching and irritation of both legs with œdema and purpura.

2. A man with generalised eczema was treated with crude tar of strength 4:32 in the morning, and in the evening of the same day headache and vomiting developed, but the skin showed no local reaction.

3. A woman suffering from dermatitis herpetiformis was treated with tar of strength 2:32, with the result that during the day headache and nausea supervened, and in the evening a macular itching eruption appeared on the arms and shoulders, and forthwith the tar applications were stopped.

Next morning the face was pale and œdematous, and on the following day the arms were swollen and tense, and the sickness was still present. Improvement after this was steady and uneventful.

White traces these cases to the fact that the drug was being collected in dirty receptacles, and the use of a clean, fresh supply was followed by no such untoward results.

Such an accident may be obviated by simply washing the tar with water, as we generally recommend.

It may also be that the use of tar in the paste form leads to greater absorption when daily applied than when used full strength, at intervals of 4 or 5 days, as is our custom. F. G.

NEW BOOKS.

Gynæcology for Students and Practitioners. By Thomas Watts Eden, M.D., F.R.C.S., F.R.C.P., and Cuthbert Lockyer, M.D., F.R.C.S., F.R.C.P. Pp. xiv. + 890. With 533 Illustrations. London: J. & A. Churchill. 1916. Price 27s. net.

There are some books which it is a real pleasure to review, and the above falls into this category. The authors have succeeded in setting forth a comprehensive account of the special diseases of women and in keeping an even balance between the pathological and clinical aspects of the subject—by no means an easy task. They have taken up a strong position on the much debated question of endometritis, and they adopt Hitschmann's and Adler's views in their entirety. They certainly make out a good case for their contention. Some admirable photomicrographs illustrate their points, and useful tables are given to assist in diagnosis and differential diagnosis.

Where the illustrations generally are so excellent, and many strikingly so, it is a pity that some of those dealing with the subject of menstruation are a little crude. Possibly war conditions may account for this.

The specific infections in women are well described, as are the general pelvic infections, which rightly include appendicitis. We agree with the authors that obstetric infections, coming as they do so frequently under the care of the gynæcologist, render it desirable that gynæcologists should have a practical knowledge of obstetrics, as gynæcology has no doubt suffered in the past from the incursions of those who are not qualified by training to understand its clinical

problems aright. The subject of ectopic pregnancy is dealt with in a masterly way. The terms internal and external capsular hæmorrhage seem, however, rather unnecessary and misleading.

We are glad to see the authors lay stress on the administration of strychnine before abdominal operations as a preventive of shock, and on the desirability of giving a hypodermic of hyoscine and morphia shortly before operative interference.

They also point out that the conventional view that abdominal operations upon the pelvic organs should not be performed during a menstrual period is a concession to sentiment, and not the expression of a surgical necessity. It is therefore unnecessary to postpone a proposed abdominal operation on account of the unexpected appearance of uterine hæmorrhage.

The statement that all three embryonic layers are *generally* to be found in so-called ovarian dermoid cysts, thus constituting them teratomata, seems hardly borne out by the usual clinical experience.

The low type of malignancy of perithelioma of the ovary is clearly accentuated.

The operative section is judiciously free from a mass of technical details, the authors rightly holding that operating cannot be learned from books. Sufficient descriptions are, however, given for the guidance of those who are accustomed to the general routine of modern surgical work.

We observe that the authors' operation of choice for retroversion of the uterus, when required, is Gilliam's operation. They do not mention that, excellent as it is in most cases, it does not appear to stand the strain of subsequent pregnancy so well as could be desired.

Curiously enough no mention is made of the administration of pituitrin in certain forms of amenorrhœa.

Enough has been said to show that this book is a valuable addition to any gynæcological library, and, taken as a whole, it is probably one of the best books on the subject of recent years.

The authors, the artists, and the publishers are to be congratulated on the production of a notable work.

Gynecology. By William Graves, A.B., M.D., F.A.C.S., Professor of Gynecology at Harvard Medical School; Surgeon-in-Chief to the Free Hospital for Women, Brookline; Consulting Physician to the Boston Lying-in Hospital. Pp. 770. With 491 Illustrations. Philadelphia and London: W. B. Saunders Co. 1916. Price 30s. net.

Professor Graves' new treatise on gynecology is a large and ambitious book, illustrated and got up generally in the superb way characteristic of the best Transatlantic medical literature. Emanating as it does from an academic chair in the medical school of Harvard,

the writing itself at once raises high expectations. It is gratifying, therefore, to be able to state that Professor Graves' work sustains the traditions of insight and erudition so long associated with his school in the minds of British readers. His writing shows a wide knowledge of his subject, alike in its practical, theoretical, and literary sides, and if one is tempted to think that he is unduly inclined to sit at the feet of the German writers as being of necessity the ultimate authorities, we must remember that America has not yet attained to that state of freedom from the Teutonic tradition to which the events of the last two years have hastened us.

One of the more novel features of the work is a series of sections dealing with the inter-relationships between gynecology and the various organs and systems of the female body. This, of course, embodies a discussion of the functions of the ductless glands, and here we fail to find any reference to the work of such observers in this country as Blair Bell, Hick, and Whitehouse. Despite omissions, however, these chapters present a useful *résumé* of our knowledge, and a stimulating reminder of our ignorance, of this fascinating and promising branch of study.

In the main portion of the book, devoted to gynecological disorders, the writer has adopted the modern pathological classification, and takes up his subjects under the headings of special and general inflammatory processes, new growths, defects of development, malpositions, injuries due to childbirth, and special gynecological diseases, such as ectopic gestation.

Like all modern authors, Professor Graves makes some endeavour to produce order out of the confusion and uncertainty surrounding the subject of "endometritis." There is no doubt that in the past thirty years this term has been as much abused in gynecology as have "eczema" in dermatology, or "influenza" and "rheumatism" in medicine. In other words, it has been used to cover inaccurate observation and loose thinking. But there is reason to believe that in the near future we shall reach some general agreement amongst gynecological pathologists as to the nature, frequency, and classification of the changes in the uterine mucosa. Meantime Professor Graves divides them into — (1) Infectious endometritis; (2) chronic interstitial endometritis, resulting from a previous infectious attack; and (3) gland hypertrophy, resulting from circulatory changes.

Under the heading of metritis the acute infection of the uterine wall is very briefly mentioned, and we have failed to find reference anywhere to the conditions commonly, even if incorrectly from a pathological point of view, grouped under one or other of the terms chronic metritis, fibrosis uteri, or subinvolution.

The last part of the book is occupied with operative work, and the author, while giving an account of most of the better-known methods

of procedure, freely and helpfully records his own preferences in regard to technique. Operations on the kidney, ureters, bladder, rectum, and even varicose veins of the leg are described, and indicate a catholic interpretation of the term "gynecology."

A special word of praise must be reserved for the illustrations, which are beautiful and worthily reproduced. The majority are the author's own handiwork, and he is to be heartily congratulated upon them. The ladies responsible for the microscopic drawings deserve an equal meed of praise for their admirable work.

Notes on Galvanism and Faradism. By E. M. Magill, M.B., B.S.(Lond.), D.P.H., R.C.S.I.(Hons.). Pp. xvi. + 220. With 67 Illustrations. London: H. K. Lewis & Co., Ltd. 1916. Price 4s. 6d. net.

This book is just what it claims to be. It is a link between the works on medical electricity proper and those written for the technical student. In it there is an earnest endeavour made to explain elementary practical details that are usually taken for granted in the more advanced works. In the preface the author states that the volume is mainly for the use of masseuses preparing for examination, but it will also be found to be of much service to the student as an introduction to a very difficult subject.

Written in a style both clear and concise, and well illustrated, this book brings the essentials of medical electricity within the grasp of those who are not disposed, or have not the leisure, to read larger books. The chapters on cells and batteries contain a mass of information not always so readily obtained, and compressed into small space. A full description of the ways in which galvanism and faradism act as therapeutic agents is followed by an account of the electrical currents used in medicine, and the sources of supply that are available. We are glad to note a lucid statement of ionic medication and the theory of ionisation, which has been, and still is, of great practical value in treating certain conditions.

Among the small books on the subject this is one of the best, and we commend it to all those whom it is designed to assist.

Principles of Diagnosis and Treatment in Heart Affections. By Sir James Mackenzie. Pp. 264. London: Henry Frowde and Hodder & Stoughton. 1913. Price 7s. 6d. net.

In this volume Sir James Mackenzie further develops his conception of heart conditions—how they may be differentiated and treated; and more particularly is he concerned with prognosis—how it may be estimated, and this is, after all, the important factor, both for the doctor and the patient. His conception of circulatory troubles, though at one time novel and original, is now being followed and recognised. It is readily understood, and appeals both to the teacher and the practitioner.

The latter in this volume receives more than encouragement to study his heart cases more thoroughly and systematically, for the author believes that on him alone depends future progress in our knowledge of heart cases; for only the practitioner sees the cases at their earliest beginnings, and he alone is able to follow their course over a period of years. And it is only by so doing that any real advances can be expected.

A particularly valuable chapter is the one dealing with such conditions as sinus irregularities, heart block, etc., more so at the present time when so many of our soldiers are returning with disabled hearts. Mackenzie has reduced these conditions to their proper significance, and has shown them to be compatible with regular work and employment, and that they are not to be looked upon as disabling and condemning a man to a sedentary and anxious future.

In the treatment of heart conditions, especially as regards the use of the so-called cardiac tonics, he pleads for a proper discrimination of their use, and lays down the conditions under which digitalis and strophanthus are really useful, and for which alone they should be used.

Anyone who in his daily work carries with him Mackenzie's conception of how heart affections should be regarded and followed will find it simplified and more interesting, and will be stimulated to study his cases in a rational manner.

ANALYTICAL REPORTS.

Kepler Cod Liver Oil with Malt Extract and Creosote.

(Burroughs Wellcome & Co.)

The difficulty of obscuring the nauseous properties of creosote has been successfully overcome in this preparation made by Messrs. Burroughs Wellcome & Co. The combination of this drug with cod-liver oil and maltine renders the preparation specially useful in cases of phthisis. As the medicament is in intimate association with the vehicle there is no tendency to separation, and the full value of the nutritive agents is obtained.

Robinson's Patent Barley and Patent Groats.

(Keen, Robinson & Co., Ltd.)

These preparations have been found specially useful in the feeding of infants. For the dilution of cows' milk, barley water made from the prepared barley adds to the nutritious value of the mixture. The barley water can be prepared in ten minutes, and is free from the impurities frequently accompanying pearl barley. The groats preparation is a pure food made from first quality oats.

Cadbury's Cocoa and Chocolate Products.

(Cadbury Brothers, Ltd.)

We have received from Messrs. Cadbury a selection of the products of the Bourneville factories. These are so well known that detailed commendation is unnecessary. The food value of high-grade cocoa and chocolate is fully recognised, and in these times it cannot be overlooked. A number of the Cadbury preparations are specially prepared and packed for transmission to the forces abroad.

Surgical Appliances.

(Robert Whitelaw.)

The frequency with which drop wrist and drop foot occur as a result of gunshot injuries has led to the production of a variety of appliances to prevent over-stretching of the paralysed muscles. Mr Whitelaw of Aberdeen has submitted to us a wrist-drop appliance and a foot-drop appliance which are at once simple and efficacious.

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MEDICAL EDUCATION IN SCOTLAND.

ACCORDING to the Regulations of the General Medical Council, a candidate for a medical qualification must (1) pass a preliminary examination; (2) register as a medical student; (3) study for five years at a recognised school; (4) obtain a degree or diploma; and (5) place his name on the *Medical Register*. No person whose name is not on the *Register* may legally sign certificates, give medical evidence in Courts, or sue for fees.

The following degrees and diplomas are available in Scotland:—Bachelor of Medicine and Bachelor of Surgery (M.B., Ch.B.), conferred by the Universities. Doctor of Medicine (M.D.) and Master of Surgery (Ch.M.) are higher qualifications conferred only on those who already hold the M.B., Ch.B.

The *Triple Qualification* (L.R.C.P.E., L.R.C.S.E., L.R.F.P.S.G.) is conferred by the two Royal Colleges and the Royal Faculty jointly. The Fellowships, Memberships, and Licences of these Corporations may also be registered as higher or additional qualifications.

Special degrees and diplomas in public health are also granted by the Universities and Corporations.

THE UNIVERSITIES.

PRELIMINARY EXAMINATION.—Before commencing his course of medical study each student shall pass a preliminary examination in (1) English, 2 Latin, 3 Mathematics, and (4) an additional language—Greek, French, German, Italian, or such other as the Senatus shall approve. In the case of a candidate whose native language is other than English, an examination in another classical language—*viz.*, Sanskrit or Arabic—may be substituted for Latin, and an examination in the candidate's native language may be substituted for the additional language. A student must pass all the subjects at not more than two examinations. A degree in Arts or Science of any recognised University exempts from the preliminary, and certain other examinations may be accepted as substitutes.

MATRICULATION.—Having passed the preliminary examination, the student must, within fifteen days after the commencement of the session, matriculate at the University and pay the fee, which is £1, 1s. for the whole year, 10s. 6d. for the summer session alone.

REGISTRATION.—Within fifteen days of commencing his studies he must register as a medical student. He must be not less than sixteen years of age, must have passed the preliminary examination, and must show that he has begun his medical studies.

THE CARNEGIE TRUST.—This Trust is prepared to pay the class fees of students (who have passed the specified preliminary examination) for all classes, whether attended within the Universities or in any of the Extra-Mural Schools. It is also prepared to pay the fees for the various special classes given by many of the Lecturers upon advanced and non-compulsory subjects. Applicants (1) must be over sixteen years of age; (2) must be of

Scottish birth or extraction, or must have given two years' attendance after the age of fourteen at a school or institution under inspection of the Scottish Education Department; and (3) must be qualified by preliminary examination under the ordinances of the Scottish Universities Commission and the regulations of the Joint Board of Examiners, to attend the classes for which payment of fees has been claimed. Schedules of application for admission to the benefit of the Trust are obtainable by written application to the Secretary of the Trust, 14 Hanover Street, Edinburgh.

UNIVERSITY OF EDINBURGH.

The University of Edinburgh offers many attractions to the student of medicine. The various departments are well equipped for purposes of teaching and research, and there are ample facilities for clinical instruction. The Royal Infirmary, the Royal Hospital for Sick Children, and the Royal Maternity Hospital are in the immediate vicinity of the University, while the Royal Asylum for the Insane, the Fever Hospital, and the various dispensaries can be reached within half an hour.

Students may attend one half of their classes in the Extramural School, and are thus offered a choice of teachers on the different subjects of the curriculum.

An important agreement between the University and the Royal Infirmary has been reached by which the teaching resources of the latter are more fully available for University students than formerly. The agreement provides that all the senior members of the Infirmary staff (those in charge of wards) become senior University lecturers and examiners, while the assistant physicians and surgeons become University lecturers, and take a share in the clinical teaching. The clinical tutors also take a recognised place in University teaching, and the medical demonstrations are held at a morning hour instead of in the evening.

Since the war broke out a very large number of students and several of the teachers have joined His Majesty's forces. The reduction in numbers has been somewhat proportionate, and with a little rearrangement the teaching has been adequately maintained.

The social side of student life is provided for in many ways. The University Union, with a membership of 1500, has a debating-hall, a library, reading rooms, billiard rooms, and a catering department.

The Royal Medical Society, founded in 1737, offers the student the advantages of an extensive medical library and reading rooms, while in its spacious hall many whose names have since become famous have made their first essay in medical debate. The Australasian Club and the South African Union are the headquarters of students from these parts of the Empire.

The Town and Gown Association provides a number of student residences, which are managed by a committee of the residents.

Women students are now admitted to the systematic lectures and practical classes within the University.

Under a recent regulation attendance on classes of instruction in the various special departments is compulsory. This change has necessitated a rearrangement of the curriculum, and an important regulation was introduced which has the effect of limiting a student's attendance on later subjects of the curriculum until he has passed the professional examinations on the earlier subjects.

Particulars regarding the curriculum will be found in the University calendar or "medical programme," published by James Thin, 55 South Bridge.

It is recommended that students begin study in the Summer Session. The curriculum is as follows :—

FOR STUDENTS BEGINNING IN SUMMER.

First Summer Term		Hours of Medical Classes
Botany		8-9
Zoology		12-1
Practical Botany	On alternate days	9-11
Practical Zoology	On alternate days	10-12
Practical Anatomy (thrice weekly)		Afternoon

(*Examinations in Botany and Zoology.*)

First Year.

Winter (1*st Term*)	
Practical Chemistry (twice weekly)	9-11
Anatomy	11-12
Chemistry	12-1
Physics[1]	1-2
Practical Anatomy	Afternoon

Winter (2*nd Term*)	
Practical Chemistry (twice weekly)	9-11
Anatomy	11-12
Chemistry	12-1
Physics[1]	1-2
Practical Anatomy	Afternoon

(*Examinations in Chemistry and Physics.*)

Summer Term	
Histology	8-10 or 11-1
Practical Anatomy.	

Second Year.

Winter (1*st Term*)	
Physiology	10-11
Practical Physiology (twice weekly)	11-1
Practical Anatomy and Demonstrations.	

Winter (2*nd Term*)—	
Physiology	10-11
Practical Physiology (twice weekly)	11-1
Practical Anatomy and Demonstrations.	

(*Examinations in Anatomy and Physiology.*)

Summer Term—	
Pathology (Practical) (thrice weekly)	8-10
Pathology (Morbid Anatomy)	10-11
Surgical Out-patients	11-12
Clinical Surgery	12-2
Practical Materia Medica	Various hours

Third Year.

Winter (1*st Term*)—	
Medicine	9-10
Pathology (Morbid Anatomy)	10-11
Clinical Medicine	11-1
Materia Medica	2-3
Pathology (including Elementary Bacteriology)	3-4

[1] The class of Physics meets thrice weekly during the Winter Session.

Winter (2nd *Term*)			Hour of Meeting of Class
Medicine			9-10
Pathology (Morbid Anatomy)			10-11
Clinical Medicine			12-1.30
Materia Medica			2-3
Pathology (including Elementary Bacteriology)			3-4

(*Examination in Pathology and Materia Medica.*)

Summer Term—			
Out-patients (Medical)			11-1
Vaccination			3-4
Dispensary Practice			Afternoon

Fourth Year.

Winter (1st *Term*)—			
Surgery			9-10
Midwifery (including Gynecology)			10-11
* Sec. A. Diseases of Skin " B. Diseases of Eye " C. Diseases of Ear, etc.	11-12	Clinical Surgery	12-2
Dispensary Practice. Infectious Diseases (once weekly) Anæsthetics.[1] Optional Classes.[2]			Afternoon

Winter (2nd *Term*)—			
Surgery			9-10
Midwifery (including Gynecology)			10-11
Sec. A. Diseases of Ear, etc. " B. Diseases of Skin " C. Diseases of Eye	11-12	Clinical Surgery	12-2
Mental Diseases (twice weekly)			3-4
Infectious Diseases (if not previously attended) Dispensary Practice Practical Midwifery			Afternoon

Summer Term—			
Operative Surgery			8-9.45
Public Health			10-11
Sec. A. Diseases of Children			11-1
" B. Diseases of Ear, etc. " C. Diseases of Skin	11-12	Clinical Medicine	12-1.30
Forensic Medicine			2-3
Infectious Diseases (if not previously attended)			Afternoon

(*Examination in Forensic Medicine and Public Health.*)

* These classes are held thrice weekly, and on remaining days students must attend Clinical Surgery at 11 o'clock.

[1] A course of instruction is given in each term.

[2] Optional courses are held in the subjects of *History of Medicine* (during the First Term of the Winter Session, twice weekly, 4 to 5 P.M., and may be attended by students who have passed the 1st Professional Examination); *Physical Methods in the Treatment of Disease* (during the First Term of the Winter Session, 4 to 5 P.M., twice weekly, and open to students who have passed the 2nd Professional Examination); *Neurology* (daily at 4 P.M. during the Second Term of the Winter Session, and open to students who have passed the 3rd Professional Examination); *Applied Anatomy* (thrice weekly, from 4 to 6 P.M. during First Term of Winter Session, and students are recommended to take the class in the Fifth Winter).

Fifth Year.

	Hour of Meeting of Class.
Winter (1*st Term*)	
Clinical Gynecology	10-11
Sec. B. Diseases of Children	11-1
„ A. Diseases of Eye 11-12 Clinical Medicine	12-1.30
„ C. Clinical Medicine or Clinical Surgery.	
Dispensary Practice } Practical Midwifery }	Afternoon
Optional Classes.	
Winter (2*nd Term*)	
Clinical Gynecology	10-11
Sec. C. Diseases of Children	11-1
Secs. A. and B. Clinical Medicine or Clinical Surgery	12-1.30
Dispensary Practice } if not previously attended Practical Midwifery }	Afternoon
Summer Term—	
Clinical Work in Hospital.	

(*Examination in Midwifery, including Gynecology, Surgery, and Medicine, and corresponding Clinical Examinations.*)

FOR STUDENTS BEGINNING IN WINTER.

First Year.

Winter (1*st Term*)	
Practical Chemistry (twice weekly)	9-11
Anatomy	11-12
Chemistry	12-1
Physics	1-2
Practical Anatomy (thrice weekly)	Afternoon
Winter (2*nd Term*)	
Practical Chemistry (twice weekly)	9-11
Anatomy	11-12
Chemistry	12-1
Physics	1-2
Practical Anatomy (thrice weekly)	Afternoon
(*Examination in Chemistry and Physics.*)	
Summer Term—	
Botany	8-9
Zoology	12-1
Practical Botany } On Alternate days {	9-11
Practical Zoology } On Alternate days {	10-12
Practical Anatomy	Afternoon
(*Examination in Botany and Zoology.*)	

Second Year.

Winter (1*st Term*)—	
Physiology	10-11
Practical Physiology (twice weekly)	11-1
Practical Anatomy.	
Winter (2*nd Term*)—	
Physiology	10-11
Practical Physiology	11-1
Practical Anatomy and Demonstrations.	

Summer Term—	Hour of Meeting of Class.
Histology	8-10 or 11-1
Practical Materia Medica.	
Practical Anatomy and Demonstrations.	

(*Examination in Anatomy and Physiology.*)

Third Year.

Winter (*1st Term*)—	
Medicine	9-10
Pathology (Morbid Anatomy)	10-11
Clinical Medicine	11-1
Materia Medica	2-3
Pathology (including Elementary Bacteriology)	3-4
Winter (*2nd Term*)—	
Medicine	9-10
Pathology (Morbid Anatomy)	10-11
Clinical Medicine	12-1.30
Materia Medica	2-3
Pathology (including Elementary Bacteriology)	3-4
Summer Term—	
Pathology (Practical) (thrice weekly)	8-10
Pathology (Morbid Anatomy)	10-11
Clinical Surgery	12-2
Practical Materia Medica (if not previously attended).	
Vaccination	3-4

(*Examination in Pathology and Materia Medica.*)

Fourth Year.

Winter (*1st Term*)—			
Surgery			9-10
Midwifery (including Gynecology)			10-11
Sec. A. Diseases of Skin „ B. Diseases of Eye „ C. Diseases of Ear, etc.	11-12	Clinical Surgery	12-2
Dispensary Practice Infectious Diseases (once weekly)			Afternoon
Anæsthetics.			
Optional Classes.			
Winter (*2nd Term*)—			
Surgery			9-10
Midwifery (including Gynecology)			10-11
Sec. A. Diseases of Ear, etc. „ B. Diseases of Skin „ C. Diseases of Eye	11-12	Clinical Surgery	12-2
Mental Diseases (twice weekly)			3-4
Infectious Diseases (if not previously attended) Dispensary Practice Practical Midwifery			Afternoon
Summer Term—			
Operative Surgery			8-9.45
Public Health			10-11
Sec. A. Diseases of Children			11-1
„ B. Diseases of Ear, etc. „ C. Diseases of Skin	11-12	Clinical Medicine	12-1.30
Forensic Medicine			2-3
Infectious Diseases (if not previously attended)			Afternoon

(*Examination in Forensic Medicine and Public Health.*)

Fifth Year.

Winter (1st Term)

	Hours of Attendance
Clinical Gynecology	10-11
Sec. B. Diseases of Children	11-1
„ A. Diseases of Eye 11-12 Clinical Medicine	12-1 3-0
„ C. Clinical Medicine or Clinical Surgery	
Dispensary Practice }	Afternoon
Practical Midwifery }	
Optional Classes.	

Winter (2nd Term)

Clinical Gynecology	10-11
Sec. C. Diseases of Children	11-1
Secs. A. and B. Clinical Medicine or Clinical Surgery	12
Dispensary Practice } if not previously attended	Afternoon
Practical Midwifery }	

Summer Term

Clinical Work in Hospital.

(*Examinations in Medicine, Surgery, Gynecology, Systematic and Medical, and Systematic and Clinical Examinations.*)

The candidate must attend Hospital for not less than three years, must attend both Clinical Medicine and Clinical Surgery for a period of at least nine months; twenty cases of Midwifery, or twelve cases and three months' attendance at a Maternity Hospital; and Post-Mortem Examinations for three months.

It is required that, before commencing the study of Practical Midwifery, every student shall have held the offices of Clinical Medical Clerk and Surgical Dresser, and have attended a Course of Lectures on Surgery and Midwifery.

Two of the five years of study must be spent at the University, and not less than eight of the compulsory subjects of study must be taken in the University.

The minimum expense of M.B. and Ch.B., including fees for Classes, Hospital, Matriculation and Examination, amounts to about £143.

Degree of M.D.

Each candidate for this degree who began medical study after 1st October 1892 must be of the age of twenty-four years or upwards, and must have obtained the degrees of M.B. and Ch.B. of the University. He must either have been engaged for two years in general practice, or for one year in the naval or military medical services, or in the medical wards of a hospital, or in scientific research. He must present a thesis written by himself on a medical subject, and pass an examination in Clinical Medicine. In this examination the candidate has to write a report and commentary on at least three cases, and has to show a practical knowledge in the application of the ophthalmoscope, laryngoscope, electrical apparatus, and sphygmograph, in the examination of the blood, and in the chemical and microscopical examination of the excreta.

The candidate who has graduated M.B. and C.M. under the old regulations may either proceed to the degree of M.D. under the old regulations (under which he is not required to pass an examination in Clinical Medicine, but must have passed examinations in Greek and in Logic or Moral Philosophy), or he may proceed to the degree as if he had graduated M.B., Ch.B. under the New Regulations.

Degree of Ch.M.

Each candidate must be not less than twenty-four years of age, must possess the degrees of M.B., Ch.B., must have attended the surgical wards of a hospital for one year, or have spent one year in scientific research or in the naval or military medical services, or two years in practice other than that restricted to medicine. He must submit a thesis on a surgical subject, and pass an examination on Clinical Surgery and its branches, Surgical Anatomy, and Operations on the Dead Body.

Fees for M.D. and Ch.M.

The fee for the M.D. degree under the old regulations is five guineas; for the M.D. or Ch.M., under the New Regulations, ten guineas. The candidate must have paid the matriculation fee for the year in which he presents himself for examination or graduation. At each reappearance for examination, under the New Regulations, the fee is five guineas.

Degrees in Public Health.

Two degrees are granted by the University of Edinburgh in the department of Sanitary Science, viz. B.Sc. and D.Sc.

Bachelor of Science.

Candidates must be graduates in Medicine of a University of the United Kingdom or of some other recognised University. In order to obtain the degree two examinations have to be passed.

First Examination.—Before entering for this examination the candidate must, after graduating in Medicine, have worked in a recognised Public Health Laboratory for eight months, of which five consecutive months must be passed in the Public Health Laboratory of the University of Edinburgh.

He must also have attended in a Scottish University a course of lectures on Physics and a course of lectures on Geology, extending over three months, and approved of by the University Court.

The subjects of examination are as follows:—

(1) *Laboratory* work—Practical, written and oral; examination of water, air, foods, beverages, condiments, sewage; soils; disinfectants; building materials; clothing; bacteriology.
(2) Physics. (3) Geology.

Second Examination.—This cannot be taken until eighteen months after graduating in medicine; nor sooner than six months after passing the First B.Sc. Examination. The candidate must have attended two separate courses on Public Health, either in the University of Edinburgh or in some other recognised University or School.

Each course must consist of forty lectures, and include Medicine in its relation to Public Health and Sanitary Engineering.

The candidate must likewise produce evidence (1) that for six months he has studied sanitary work under a Medical Officer of Health for a county or burgh of not less than 25,000 inhabitants; (2) that he has studied clinically for three months infectious diseases in a recognised institution; (3) that for three months he has been instructed by a recognised teacher in mensuration and drawing.

The subjects of examination are:—

(1) Sanitation; (2) Sanitary Law; (3) Vital Statistics; (4) Medicine in Relation to Public Health.

The candidate is examined orally, practically, and by a written paper. *Sanitation* includes making reports on dwellings, workshops, hospitals and sanitary schemes.

The University Court may modify the work and instruction prescribed from time to time.

DOCTOR OF SCIENCE.

A graduate after having held the degree of B.Sc. for five years may present himself for the D.Sc. He must present a thesis or a published work or memoirs, the result of his own research, and must pass an examination in Public Health, and in such of its special subjects as the Senatus may determine. The candidate must submit the subject in which he proposes to be examined for approval not less than two months before the examination.

FEES PAYABLE. First and second examinations, £3, 3s. each; for D.Sc., £10, 10s.

INSTITUTIONS FOR CLINICAL INSTRUCTION IN EDINBURGH.

Royal Infirmary. 921 beds and 42 cots. Fees: perpetual ticket, £12; one year, £6, 6s.; six months, £4, 4s.; three months, £2, 2s. Clinical instruction is given daily in Medicine, Surgery, and all their special branches.

Royal Hospital for Sick Children. 120 beds. Hospital ticket, £1, 1s. Fee for Qualifying Course, £2, 2s.

City Hospital for Infectious Diseases. 600 beds. Fee, £1, 1s.

Royal Maternity and Simpson Memorial Hospital. 40 beds. The Maternity Residency affords accommodation for twelve students.

Royal Asylum, Morningside. 500 beds.

The fee for a qualifying course at each of these last two institutions is £2, 2s.

Royal Victoria Hospital for Consumption and Diseases of the Chest. 50 beds. Out-Patient Department at Spittal Street.

Eye, Ear and Throat Infirmary. 6 beds; 2700 Out-Patients yearly. Fee for three months, £1, 1s.

Royal, New Town, Medical Missionary (Cowgate), Western, Provident Marshall Street, Eye, and Skin Dispensaries.

PROFESSORS AND LECTURERS IN EDINBURGH.

The Courses given by the Extramural Lecturers are recognised by the University and other examining boards as qualifying for graduation.

Botany— Professor Bayley Balfour, M.D., Botanical Gardens.
James A. Terras, B.Sc., New School.

Zoology— Professor J. Cossar Ewart, M.D., University.
Malcolm Laurie, D.Sc., Surgeons' Hall.
Marion I. Newbigin, D.Sc., Surgeons' Hall.
Hugh Miller, F.Z.S., 29 George Square.

Biology— Malcolm Laurie, D.Sc., Surgeons' Hall.
Marion I. Newbigin, D.Sc., Surgeons' Hall.
C. R. Whittaker, F.R.C.S., New School.

Physics— Professor C. G. Barkla, D.Sc., University.
C. G. Knott, D.Sc., University.
Dawson Turner, M.D., Surgeons' Hall.
A. M'Kendrick, F.R.C.S., New School.

Chemistry— Professor Walker, University.
G. H. Gemmell, F.I.C., 4 Lindsay Place.
T. W. Drinkwater, Ph.D., Surgeons' Hall.

Anatomy— Professor A. Robinson, M.D., University.
J. Ryland Whitaker, M.B., Surgeons' Hall.

Applied Anatomy
H. J. Stiles, M.B., University.
J. Ryland Whitaker, M.B., Surgeons' Hall.

Physiology Professor Sir E. A. Schafer, LL.D., University.
Alexander Goodall, M.D., Surgeons' Hall.

Materia Medica and Therapeutics—
Professor Sir Thomas R. Fraser, M.D., University.
William Craig, M.D., Surgeons' Hall.
John Orr, M.D., New School.

Pathology— Professor Lorrain Smith, M.D., University.
James Miller, M.D., Surgeons' Hall.

Surgery— Professor Alexis Thomson, *C.M.G.*, University.
A. A. Scot Skirving, *C.M.G.*, F.R.C.S., 27 Nicolson Square.
G. L. Chiene, F.R.C.S., New School.
Henry Wade, F.R.C.S., Surgeons' Hall.
J. W. Struthers, F.R.C.S., New School.
W. J. Stuart, F.R.C.S., 59 Forrest Road.
Lewis Beesly, F.R.C.S., Surgeons' Hall.

Clinical Surgery—
The Surgeons of the Royal Infirmary.
Professor F. M. Caird.
Professor Alexis Thomson.
J. W. B. Hodsdon.
David Wallace.
Alexander Miles.
John W. Dowden.
A. A. Scot Skirving.
And Assistant Surgeons.

Practice of Medicine—
Professor G. Lovell Gulland, M.D., University.
Harry Rainy, M.D., 27 Nicolson Square.
R. A. Fleming, M.D., Surgeons' Hall
A. Dingwall Fordyce, M.D., Dental Hospital.
W. T. Ritchie, M.D., Surgeons' Hall.

Clinical Medicine
The Physicians of the Royal Infirmary.
Professor Sir Thomas R. Fraser.
Professor G. Lovell Gulland.
Professor Wm. Russell.
Sir R. W. Philip.
Dr. Graham Brown.
Dr. F. D. Boyd.
Dr. R. A. Fleming.
Dr. Harry Rainy.
And Assistant Physicians.

Midwifery and Gynecology—
Professor Sir J. Halliday Croom, M.D., University.
D. Berry Hart, M.D., Surgeons' Hall.
J. W. Ballantyne, M.D., Surgeons' Hall.
A. H. F. Barbour, M.D., University and Royal Infirmary.
J. Haig Ferguson, M.D., New School.
N. T. Brewis, F.R.C.S., Royal Infirmary.
G. F. B. Simpson, M.D., New School.
E. M. Inglis, M.B.
John McGibbon, M.B., 59 Forrest Road.
H. S. Davidson, F.R.C.S., Nicolson Square.

Insanity— G. M. Robertson, M.D., University and Royal Asylum.
John Keay, M.D., Surgeons' Hall and Bangour Village Asylum

Diseases of the Eye—
W. G. Syme, M.D., Royal Infirmary.
J. V. Paterson, M.B., Royal Infirmary.
A. H. H. Sinclair, M.D. (Ophthalmoscopy), 6 Lauriston Place.

Vaccination—W. G. Aitchison Robertson, M.D., D.Sc., Royal Dispensary.
W. D. D. Small, M.B., Provident Dispensary.

Diseases of Children—
John Thomson, M.D.
H. J. Stiles, M.B.
J. S. Fowler, M.D.
J. W. Simpson, M.D.

Diseases of the Skin—
Norman Walker, M.D., Royal Infirmary.
Frederick Gardiner, M.D., Surgeons' Hall.

Diseases of the Ear, Nose, and Throat—
A. Logan Turner, M.D., Royal Infirmary.
J. Macdonald Fitzpatrick, M.B., Royal Infirmary.
J. S. Fraser, M.B., Surgeons' Hall.

Forensic Medicine—
Professor Harvey Littlejohn, F.R.C.S., University.
W. G. Aitchison Robertson, M.D., D.Sc., Surgeons' Hall.

Public Health—
Professor C. Hunter Stewart, M.B., University.
W. G. Aitchison Robertson, M.D., D.Sc., Surgeons' Hall.
Wm. Robertson, M.D., Surgeons' Hall.

Fevers— Alexander James, M.D., City Hospital.
C. B. Ker, M.D., City Hospital.

Bacteriology—
Professor James Ritchie, M.D., University.
J. Taylor Grant, M.D., 4 Lindsay Place.
James Miller, M.D., Surgeons' Hall.

Diseases of Tropical Climates—
Major D. G. Marshall, I.M.S., University and Surgeons' Hall.

Practical Medicine and Physical Diagnosis—
Alexander Goodall, M.D., Surgeons' Hall.
R. A. Fleming, M.D., Nicolson Square.
J. D. Comrie, M.D., 50 Forrest Road.

Neurology—J. J. Graham Brown, M.D., University.

Physical Methods in the Treatment of Disease—
Harry Rainy, M.D., University.

Diseases of the Chest—
Sir R. W. Philip, M.D., Royal Victoria Hospital.

Medical Electricity and Radiography—
Dawson Turner, M.D., Surgeons' Hall.

Practical Anæsthetics—
J. Stuart Ross, M.B., University.

History of Medicine—
J. D. Comrie, M.D., University.

UNIVERSITY OF GLASGOW.

Degree of M.B. and Ch.B.

Within recent years the facilities for both scientific and practical training have been much extended and improved. New and fully equipped laboratories have been added in connection with nearly all the scientific subjects, the most recent addition being a large building, completed at a cost of £40,000, for the departments of Physiology, Materia Medica, and Public Health. There is a large and well-equipped Pathological Institute at the Western Infirmary in which the University Classrooms are placed, and the Professor of Pathology is *ex officio* Pathologist to the Infirmary, and has control of all the pathological material for purposes of instruction and investigation. A corresponding arrangement forms part of the new scheme recently completed with regard to the Royal Infirmary. The Western Infirmary is close to the University, and has hitherto been the chief field of clinical instruction of University students. A scheme has, however, been carried through, according to which University Chairs, on the same footing as those already in existence, have been instituted at the Royal Infirmary in the subjects of Pathology, Medicine, Surgery, Midwifery, and Gynecology. Students accordingly have the option of taking the subjects of the two final years of study at the Royal Infirmary, and in this way the advantage is afforded of a very wide clinical field along with systematic instruction under University Professors. The great disadvantage of attending classes at Gilmorehill and going to the Royal Infirmary, at a considerable distance, for clinical work is thus done away with.

The latest development is the institution, at the Western Infirmary, of a Laboratory for Clinical Pathology, the Director of which is also a University Lecturer, and gives instruction to University students in the scientific methods of clinical diagnosis.

Under the New Ordinance of the University Court, which came into operation on 1st October 1911, the regulations for these Degrees (except in regard to the Preliminary Examination) have been considerably altered, the chief modifications being as follows: 1. A rearrangement of the subjects of the four Professional Examinations. 2. The rendering compulsory of some courses which hitherto have been optional. 3. The imposition of restrictions as to the period at which certain subjects of the curriculum can be taken.

The academical year is now divided into three terms of about ten teaching weeks each, and the following list gives the subjects of the several Professional Examinations, with the period of study required :—

First Examination.

Chemistry (including Organic Chemistry), 2 terms; with Practical Chemistry 1 term.
Physics (with practical work), 1 term.
Botany (with practical work), 1 term.
Zoology (with practical work), 1 term.

Second Examination.

Anatomy and Practical Anatomy, 5 terms.
Physiology and Practical Physiology, 3 terms.

Third Examination.

Materia Medica and Therapeutics, 2 terms.
Pathology and Practical Pathology, 3 terms.

FOURTH EXAMINATION.

Medical Jurisprudence and Public Health, 2 terms.
Surgery, 2 terms.
Practice of Medicine, 2 terms.
Midwifery and Diseases Peculiar to Women and Infants, 2 terms.

The candidate must have attended the Medical and Surgical practice of a general hospital for three years, and courses of Clinical Surgery and Clinical Medicine of nine months in each case. He must also have received instruction, under conditions laid down, in the following subjects:—

Mental Diseases.
Practical Pharmacy.
Out-Patient Practice.
Clinical Clerking in Medicine.
Clinical Clerking or Dressing in Surgery.
Post-Mortem Examinations.
Infectious Diseases.
Gynecology.
Diseases of Children.
Ophthalmology.
Diseases of the Ear and Throat.
Dermatology.
Practical Midwifery with the Conduct of Cases of Labour.
Vaccination.
Administration of Anæsthetics.
Operative Surgery.

The following courses cannot be taken till after the end of the terms of the curriculum indicated in each case:—

Physiology and Practical Physiology—third term, and not (except Practical Histology) till at least three of the subjects of the First Examination have been passed.

Materia Medica and Therapeutics, and Pathology and Practical Pathology—sixth term.

Medical Jurisprudence and Public Health—after completion of classes for the Third Professional Examination.

Midwifery, etc., Surgery and Medicine—ninth term, with the exception that Surgery may be attended after the sixth term, provided that the candidate has passed the Second Professional Examination.

Hospital Practice, Clinical Medicine, Clinical Surgery—sixth term.

The curriculum extends over five years, two of which must be spent in the University of Glasgow. The remaining three years may be spent elsewhere, as indicated in the Ordinance and under the conditions thereby imposed.

Except in the case of Medicine, Surgery, and Midwifery, the Senate may accept the Professional Examinations of other Scottish Universities.

There are a number of other administrative regulations which need not here be specified in detail.

The examination fees are £23, 2s. in all, with an additional fee of £1, 1s. for every re-entry. The cost of the curriculum amounts roughly to £145, spread over the five years of the course, and at present the class fees are charged at so much a class. There is, however, a movement on foot to introduce a "composition" or "inclusive" fee per session, but the total will work out at practically the above figure.

CLINICAL FACILITIES.

The following general hospitals, all of which are equipped in a modern fashion, are available for instruction of University students, viz. the Western Infirmary close to the University, the Royal Infirmary, to which the new

Medical Chairs are attached, each of these having at present about 600 beds, and the Victoria Infirmary, with 260 beds, on the south side of the city.

The Eye Infirmaries at 174 Berkeley Street and 80 Charlotte Street (between them 100 beds), and the Ophthalmic Institution at 126 West Regent Street (35 beds), furnish ample opportunities for instruction in the important branch with which they deal; Insanity is equally well provided for at Gartnavel (460 beds), at Hawkhead (700 beds), at Gartloch (806 beds), and at Woodilee (1160 beds), while the City Fever Hospitals at Ruchill (540 beds) and Belvidere (680 beds) are available for the study of Zymotic Diseases. The Ear, the Throat and Nose, and the Skin are dealt with in the Western and Royal Infirmaries.

A new Maternity Hospital, with every modern convenience and equipment, was recently opened in Rottenrow, with accommodation for 104 patients, and a Hospital for Sick Children, of greatly increased dimensions, in freer air, has been erected at Yorkhill within a short distance of the University. The beds number 200.

The Ordinance is applicable alike to men and women students, and much of the instruction is given in "mixed" classes by the Professors. There are, however, exceptions to this, some classes for women alone being held in a separate building (Queen Margaret College), and some for both sexes (in the main buildings at Gilmorehill) at different hours. The Hospital work in the case of women has hitherto been taken entirely in the Royal Infirmary, there being no accommodation for them in the Western. This arrangement is likely to continue.

PROFESSORS.

Zoology— Professor Graham Kerr, M.A., F.R.S.

Chemistry— (Vacant).

Practical Physics—
Professor Gray, M.A., LL.D., F.R.S.

Botany— Professor Bower, Sc.D., F.R.S.

Anatomy— Professor Bryce, M.A., M.D.

Physiology— Professor Noel Paton, B.Sc., M.D., F.R.S.

Materia Medica and Therapeutics—
Professor Stockman, M.D.

Pathology— Professor Muir, M.A., M.D., F.R.S.

Medical Jurisprudence—
Professor Glaister, M.D., D.P.H.(Camb.).

Surgery and Clinical Surgery—
Professor Sir William Macewen, M.D., LL.D., D.Sc., F.R.S.

Midwifery— Professor Murdoch Cameron, M.D.

Practice of Medicine and Clinical Medicine—
Professor T. Kirkpatrick Monro, M.A., M.D.

Public Health—
Professor Glaister, M.D., D.P.H.(Camb.).

Pathology— Professor John H. Teacher, M.A., M.D.

Medicine and Clinical Medicine—
Professor Walter K. Hunter, D.Sc., M.D.

Surgery and Clinical Surgery—
Professor Robert Kennedy, M.A., D.Sc., M.D.

Midwifery—
Professor John M. Munro Kerr, M.D.

LECTURERS.

Organic Chemistry—
T. S. Patterson, Ph.D., D.Sc.

Physiological Chemistry—Vacancy.

Psychology and Philosophy—
Henry J. Watt, M.A., Ph.D., D.Phil.

Ear—Thomas Barr, M.D., and J. Kerr Love, M.D.

Throat and Nose—
James Walker Downie, M.B., and J. Macintyre, M.B.

Skin—John Wyllie Nicol, M.B., and G. McIntyre, M.B.

Clinical Pathology—
John Shaw Dunn, M.A., M.D.

Bacteriology—
W. B. M. Martin, M.D.

Pathological Histology—
G. Haswell Wilson, M.B., Ch.B.

Physics—James G. Gray, D.Sc.

Embryology—
James F. Gemmill, M.A., D.Sc., M.D.

Insanity—Landel R. Oswald, M.B., and J. H. Macdonald, M.B.

DEAN.

Professor T. H. Bryce, M.A., M.D.

DEGREE OF M.D.

This degree is open to holders of the M.B., Ch.B. diploma, after a period of one or two years, according to circumstances, has elapsed since the date of the latter. The requirements are (1) an Examination in Clinical Medicine, or in some approved department of Medical Science or Practice; (2) a Thesis on any branch of knowledge comprised in the examinations for M.B., Ch.B., excepting a subject which is exclusively surgical; and (3) a fee of £15, 15s., with an extra charge of £5, 5s. for each re-entry.

DEGREE OF CH.M.

This may be obtained on practically the same terms as the M.D. degree, the only differences being (1) that the examination is on Surgical Anatomy, operations upon the dead body, on Clinical Surgery or an approved special department of Surgery, and (2) that the Thesis must not be on a subject which is exclusively medical.

DEGREE OF B.SC. IN PUBLIC HEALTH.

Candidates must be graduates in Medicine of a University in the United Kingdom or of some other University recognised for the purpose by the Glasgow University Court, and they must thereafter have received practical instruction, including instruction in Chemistry, Bacteriology, and the Pathology of the Diseases of Animals transmissible to man, for at least twenty hours per week during a minimum period of eight months, five consecutive months of which must be in the Public Health Laboratory of the University of Glasgow. Either before or after graduation in Medicine they must also have attended, in the University of Glasgow or elsewhere, courses of Physics and Geology, an after graduation two separate courses in Public Health (Medicine and Engineering), as well as practically studying sanitary work for six months

under a Medical Officer of Health in the United Kingdom, or a Sanitary Staff Officer of Health of the Royal Army Medical Corps, besides attending three months' practice of a Hospital for Infectious Diseases, where methods of administration can be studied, and three months in Mensuration and Drawing. The examinations are, *First*, Public Health, Laboratory Work, Physics, and Geology; *Second*, Sanitation, Sanitary Law, Vital Statistics, and Medicine in its bearings on Public Health. The examination fee is £6, 6s.

Degree of D.Sc. in Public Health.

Five years after obtaining the B.Sc. degree, graduates may proceed to the higher Degree of D.Sc., the requirements being (*a*) a Thesis or a published memoir or work to be approved by the Senate; and (*b*) an examination in Public Health and in such of its special departments as the Senate and University Court may determine. The fee for this degree is £10, 10s.

QUEEN MARGARET COLLEGE FOR WOMEN.

A full course of Medicine and Surgery is obtainable partly at Queen Margaret College, but in recent years the tendency has been to have mixed classes at Gilmorehill. The regulations, fees, etc., are similar to those for men. The buildings are pleasantly situated in grounds of their own, close to the Botanic Gardens. The anatomical department is excellently arranged and most complete. Clinical work is amply provided for in the Royal Infirmary and its Dispensaries, and in the Royal Hospital for Sick Children, the Glasgow Maternity Hospital, the Royal Asylum of Gartnavel, and the Belvidere Fever Hospital. There are also arrangements for special study and research.

Students can have board and lodging at Queen Margaret Hall, within easy reach of the College, at the rate of about one guinea per week.

All information necessary can be obtained from Miss Melville, Queen Margaret College, Glasgow.

ST. MUNGO'S COLLEGE.

This, the Medical School of the Royal Infirmary, the largest hospital in Glasgow, is situated in Cathedral Square, Castle Street, and has car communication with every part of the city. St. Mungo's College is in the Infirmary grounds.

The Infirmary has, including the Ophthalmic Department, over 620 beds, the average number occupied in 1912 being 620. When the reconstruction of the Infirmary, long in progress, is completed, it will have about 700 beds. There are special beds and wards for Diseases of Women, of the Throat, Nose and Ear, Venereal Diseases, Burns, and Septic Cases.

At the Outdoor Department the attendances in 1905 numbered over 62,000. In addition to the large Medical and Surgical Departments, there are Departments for Special Diseases—namely, Diseases of Women, of the Throat and Nose, of the Ear, of the Eye, of the Skin, and of the Teeth. A fully-equipped Electrical Pavilion was opened a few years ago, and year by year the latest and most approved apparatus for diagnosis and treatment has been added. Wards are set apart for the teaching of women students.

Appointments.—Five House-Physicians and nine House-Surgeons, having a legal qualification in Medicine and Surgery, who board in the Hospital free of charge, are appointed every six months. Clerks and Dressers are appointed by the Physicians and Surgeons. As a large number of cases of acute diseases and accidents of a varied character are received, these appointments are very valuable and desirable.

Fees.—The fees for hospital attendance, including Clinical Lectures and

Tutorial Instruction, attendance at the Outdoor Department, at the Pathological Department, Post Mortem Examinations, and the use of the Museum, which has not long since been rearranged and catalogued, are as follows:—For one year, £10, 10s.; for six months, £6, 6s.; for three months, £4, 4s. Students who have paid fees to the amount of £21 to the Glasgow Royal Infirmary are permitted to attend, in any subsequent year or years, one Winter and one Summer Course of Instruction in the Infirmary without further payment; and students who have paid to any other hospital in the United Kingdom fees, being not less than £21, in virtue of which they are entitled to attend without further payment, shall be admitted as students of the Royal Infirmary on payment of £3, 3s. for six months, or £1, 11s. 6d. for three months.

THE ANDERSON COLLEGE OF MEDICINE,

DUMBARTON ROAD, PARTICK, GLASGOW.

The old Institution known as "Anderson's University" was founded by the will of John Anderson, M.A., F.R.S., in 1795, and the medical school connected therewith dates back to the year 1799.

In 1877 the name of the Institution was altered from "Anderson's University" to "Anderson's College." In 1887 the medical school of Anderson's College became a distinct Institution known as "Anderson's College Medical School."

The new buildings are situated in Dumbarton Road, adjoining the Western Infirmary and the University. They are constructed on the best modern principles, and are provided with all the appliances requisite for the conduct and management of a fully-equipped medical school.

Classes are conducted in all the subjects of the five years' curriculum:—

Anatomy—Vacant.
Physics—Professor Peter Bennett.
Chemistry—Professor J. Robertson Watson, M.A.
Botany—Professor B. G. Cormack, M.A., B.Sc.
Zoology—Professor W. Ferguson Mackenzie, M.B.
Physiology—Professor Hugh Morton, M.D.
Materia Medica—Professor J. P. Duncan, M.B., B.Sc.
Medical Jurisprudence—Professor Carstairs Douglas, D.Sc., M.D., F.R.S.E.
Midwifery—Professor W. D. Macfarlane, M.B., C.M.
Surgery—Professor Archibald Young, B.Sc., M.B.
Practice of Medicine—Professor John Cowan, B.A., D.Sc., M.D.
Ophthalmic Medicine and Surgery—Professor A. J. Ballantyne, M.D.
Aural Surgery—Professor James Galbraith Connal, M.D.
Diseases of Throat and Nose—Professor John Macintyre, M.B., F.R.S.E.
Mental Diseases—Ivv Mackenzie, M.A., B.Sc., M.D.
Public Health Laboratory—Professor Carstairs Douglas, D.Sc., M.D., F.R.S.E.
Pathology—At the Western or Royal Infirmary.
Diseases of the Skin—J. Goodwin Tomkinson, M.D.
Dental Anatomy and Physiology—W. Wallace, M.A., M.D., L.D.S.
Dental Surgery and Pathology—John Watt, L.R.C.P., L.D.S.
Dental Metallurgy—Chas. Read, L.D.S.
Dental Bacteriology—Professor Carstairs Douglas, M.D., D.Sc., F.R.S.E.

Degrees and Diplomas.—Certificates of attendance on the classes at Anderson's College Medical School are received by the Universities of London and Durham, by the Royal University of Ireland, and by all the Royal Colleges and Licensing Boards in the United Kingdom. They are also recognised by the Universities of Glasgow and Edinburgh under certain conditions which are stated in the Calendar of this school. The Public Health Laboratory Course is recognised as qualifying for the Diploma granted by the Universities of Oxford, London, and Cambridge, the Scottish Conjoint Board, and the Royal Irish Colleges.

Candidates for the Licence in Dental Surgery can obtain at this School the full medical and dental curriculum which is required. The clinical work special to dentistry is conducted at the Glasgow Dental School, 15 Dalhousie Street.

Malcolm Kerr Bursary in Anatomy. Value about £10. Open to students of the junior anatomy class.

The Carnegie Trust will pay the fees of students at Anderson's, on conditions regarding which particulars may be obtained from Sir W. S. M'Cormick, LL.D., Carnegie Trust Offices, Edinburgh.

Class Fees.—For each course of lectures (anatomy, ophthalmic medicine and surgery, aural surgery, diseases of throat and nose, mental diseases, diseases of the skin, and dental classes excepted): first session, £2, 2s.; second session (in Anderson's College), £1, 1s.; afterwards free. Lectures on ophthalmic medicine and surgery, aural surgery, diseases of the throat and nose, mental diseases and diseases of the skin, each course, £1, 1s. For practical classes (except anatomy), viz. chemistry, botany, zoology, physiology, pharmacy, operative surgery: first session, £2, 2s.; second session, £2, 2s.

Reduced joint fees in zoology and in botany, for lectures and practical class when taken in same summer session, £3, 3s.; for either course separately, £2, 2s.

Anatomy.—Winter—first session, lectures and practical anatomy, £5, 5s.; practical anatomy alone, £2, 2s.; second session, lectures and practical anatomy, £4, 4s.; practical anatomy, £2, 2s.; for summer fees, see Calendar.

Dental Classes.—£3, 3s. each.

ABERDEEN UNIVERSITY.

The course of study for the degree of M.B., Ch.B. extends over five years, of which two at least must be spent in the University of Aberdeen.

The curriculum is the same as in the other Scottish Universities as far as relates to attendance on University classes, to clinical study at a General Hospital, to attendance on courses of Clinical Surgery, Clinical Medicine, Mental Diseases, and Practical Pharmacy, Operative Surgery, Anæsthetics, to instruction in Vaccination, to attendance on Cases of Labour, and to the practice of a Dispensary.

The candidate must also, before admission to the final examination, produce the following certificates:—

1. That he has been present at not fewer than twenty-five post-mortem examinations, some of which he must have personally taken part in performing.

2. That he has attended a course of instruction in Infectious Diseases consisting of not fewer than ten meetings, in a Hospital for the treatment of such diseases containing at least a hundred beds.

3. That he has attended in a Hospital a course of instruction in Gynæcology consisting of not fewer than twenty meetings.

4. That he has attended in a special hospital a course of instruction in the Diseases of Children, consisting of not fewer than twenty meetings.

5. That he has attended in the Ophthalmological Department of a Hospital or Dispensary a course of instruction in Ophthalmology, consisting of not fewer than thirty meetings extending over one term.

6. That he has attended in a Public Hospital or Dispensary a course of instruction in Diseases of the Ear, Nose, and Throat, consisting of not fewer than twenty meetings.

7. That he has attended in a Public Hospital or Dispensary a course of instruction in Dermatology, consisting of not fewer than twenty meetings.

Certificates for these various classes and courses must attest not only regular attendance, but also due performance of the work.

There is no prescribed order of study, but a scheme, representing the minimum curriculum, has been drawn up for the guidance of students, and is printed in the Calendar.

THE FOLLOWING ARE THE CLASSES IN THE MEDICAL FACULTY:—

WINTER SESSION.

Zoology—Professor John Arthur Thomson, M.A., LL.D.
Chemistry (Senior and Junior)—Professor Frederick Soddy, M.A., F.R.S., and Assistants.
Anatomy—Professor Reid, M.D., F.R.C.S.
Practical Anatomy—Professor Reid and Assistants.
Physiology (Senior and Junior)—Professor Mac William, M.D., F.R.S.
Materia Medica—Professor Cash, M.D., LL.D., F.R.S.
Pathology (Senior and Junior)—Professor Theodore Shennan, M.D., F.R.C.S.E.
Public Health—Mr. John Patrick Kinloch, M.D., *Lecturer*.
Surgery—Professor John Marnoch, C.V.O., M.A., M.B., C.M.
Midwifery—Professor Mackintosh, M.A., M.D.
Medicine and Diseases of Women and Children—Professor R. G. M'Kerron, M.A., M.D.

SUMMER SESSION.

Botany—Professor Trail, M.A., M.D., F.R.S.
Practical Botany—Professor Trail.
Zoology—Professor Thomson.
Practical Zoology—Professor Thomson.
Physics—Professor Niven, M.A., D.Sc., F.R.S.
Practical Physics—Professor Reid and Assistants.
Practical Materia Medica and Pharmacy—Professor Cash and Assistants.
Physiology (Senior and Junior)—Professor Mac William.
Forensic Medicine—Professor Hay, M.D., LL.D.
Practical Histology and Forensic Medicine—Professor Hay.
Pathology (Senior and Junior)—Professor Shennan.
Practical Medicine and Clinical Medicine and Clinical Diseases of Children—Professor M'Kerron.

Systematic and Practical Pathology, and Systematic and Practical Physiology respectively, are taught as part of one continuous course, occupying a whole calendar year, i.e. a summer and a winter session.

There are Assistants to the Professors in the Medical Faculty appointed annually, three in the Department of Chemistry, two in the Departments of Anatomy, Physiology, Botany, Zoology, Pathology, and Materia Medica, and one in each of the other departments.

Clinical Medicine and Clinical Surgery are taught by the Physicians and Surgeons of the Royal Infirmary.

The following are recognised as Lecturers:—

Lecturer on Mental Diseases	.	Wm. Reid, M.D.
,, Ophthalmology	.	C. H. Usher, M.B., B.S., F.R.C.S. A. Rudolph Culloway, M.A., M.B., C.M.
,, Vaccination	.	T. Fraser, M.A., M.B., Ch.B.
,, Skin Diseases	.	J. F. Christie, M.A., M.B., C.M.
,, Diseases of Ear, Throat, and Nose	.	J. Mackenzie Booth, M.D., C.M. H. Peterkin, M.B.
,, Medical Electricity	.	J. R. Levack, M.B., C.M.
,, Anæsthetics	.	Alex. Ogston, M.B., C.M. D. W. Geddie, M.B., C.M.

All the University Classes are held at Marischal College.

Tutorial Classes are held in connection with most of the Systematic Courses, and practical instruction is given in the fully-equipped Laboratories connected with the several departments.

Graduates or others desirous of engaging in special study or research may be allowed by the Senatus to work in any of the Laboratories on payment of the usual matriculation fee.

General clinical instruction is obtained in the following Medical Institutions :—

The Royal Infirmary of Aberdeen.

This General Hospital, situated about seven minutes' walk from Marischal College, has been recently constructed on the most modern principles, and is fully equipped with all the requirements for medical work and teaching. It accommodates upwards of 270 patients : the number of patients admitted during the year 1914 was 3242, and the number of out-patients treated during the same period was 15,214.

Six resident medical officers are appointed annually, three in May and three in September, to hold office for twelve months. Salary, £26, 5s. with board.

Fees.—Perpetual fee to hospital practice, £10, or first year, £5, 10s., second year, £5, afterwards free ; clerkship in medicine, £1, 1s. ; dressership in surgery, £1, 1s. ; pathological demonstrations, £2, 2s. (Special courses of lectures are charged for.)

The Royal Hospital for Sick Children.

Is situated about five minutes' walk from Marischal College, and accommodates over 80 patients. The number of patients admitted in 1914 was 888, and the number of out-patients treated was 2057. Each student must act as clerk for six weeks in the medical and surgical wards respectively.

There are two resident medical officers, senior and junior, who hold office for six months. Salary at the rate of £25 per annum in the case of the senior resident, and £20 per annum in the case of the junior resident.

Fee for hospital practice, £2, 2s. first year.

Note.—The above Hospital is temporarily removed to Kepplestone House.

The Royal Asylum.

Is about fifteen minutes' walk from Marischal College. It accommodates over 950 patients, and has been recently fitted up with a fully-equipped hospital and a laboratory.

The senior physician is recognised by the University as lecturer on mental diseases, and delivers a qualifying course of lectures.

The City (Fever) Hospital.

Is about ten minutes' walk from Marischal College, and accommodates 250 patients.

Senior students are admitted for instruction in fevers twice a week under the visiting physician (who is the Medical Officer of Health for the City) and his assistant. Fee, £1, 1s.

General Dispensary and Vaccine Institution.

This is about five minutes' walk from Marischal College.

The total number of cases treated during 1914 was 6088, and the number of patients treated at their own homes 1085.

Fees.—General practice, £3, 3s. ; vaccination certificate and instruction, £1, 1s.

Aberdeen Maternity Hospital.

This Institution is situated at the top of Castle Terrace, less than ten minutes' walk from Marischal College. Contains 18 beds. The number of patients treated in the hospital during 1914 was 203 and at their own homes 96—in all, 299. Fee, £3, 3s.

Ophthalmic Institution.

This Institution is situated about three minutes' walk from Marischal College. The surgeon in charge is recognised by the University as a lecturer on ophthalmology.

During 1914, 33 in-patients and 3080 out-patients were treated.

PROFESSIONAL EXAMINATIONS.

There are four examinations: the subjects and regulations of these are common to the Universities of Aberdeen and Glasgow.

Degree of M.D.

The regulations with regard to the age and other qualifications of the candidate are similar to those in the other Scottish Universities. He must submit a thesis written by himself upon any medical subject, and pass an examination in Clinical Medicine or in some Special Department of Medical Science or Practice.

Degree of Ch.M.

Each candidate must be not less than twenty-four years of age, and must hold the degree of M.B., Ch.B. of the University. He must produce a certificate of having been engaged for at least one year in attendance in the surgical wards of a hospital, or in scientific research, or in the naval and military services, or for two years in practice other than practice restricted to medicine. He must present a thesis on a surgical subject and pass an examination on Clinical Surgery, Surgical Anatomy, and Operations on the Dead Body.

Diploma in Public Health (D.P.H.).

The diploma is conferred, after special instruction and examination, on any one who has been at least twelve months a graduate in medicine of a University in the United Kingdom; if not a graduate of Aberdeen University, the candidate must attend a course of instruction in this University in one or more subjects embraced in the examination for the diploma.

Each candidate must have attended a course of instruction in Public Health.

The qualifying post-graduate instruction embraces—

(*a*) Regular attendance, for three months, at a hospital for infectious diseases, at which opportunities are afforded for the study of methods of administration.

(*b*) Daily association for a period of six months (of which at least three months must be distinct from the period of laboratory instruction) in the duty, routine and special, of Public Health Administration, under the supervision of a recognised Medical Officer of Health.

(*c*) Practical instruction, for at least six months, in laboratory work, which includes examination of water, air, soil and foods, and the study of bacteriology, disinfection, ventilation, water supply and sewerage, and the framing of reports of analysis. The laboratory attendance must extend over at least fifteen hours a week.

The qualifying courses of laboratory instruction in Aberdeen University are given in the Public Health Laboratory (fee, £6, 6s.), and the Bacteriological Laboratory (fee, £4, 4s.).

Instruction in Public Health Administration is given by Professor Hay, Medical Officer of Health of the City of Aberdeen (fee, £6).

Instruction is given in the Drawing and Interpretation of Plans (fee, £1, 1s.).

A short course of lectures is given on Statistical Methods and their application to Public Health.

The diploma is conferred after an examination in March and July of each year.

The examination is written, oral and practical, and is divided into two parts.

Part I. embraces the following subjects in their application to Public Health :—

(*a*) Physics, Engineering, and Meteorology.
(*b*) Chemistry, Microscopy, and Bacteriology.

Part II. embraces

(*a*) General Hygiene.
(*b*) Sanitary Law and Vital Statistics.

Part I. may be taken alone, or both parts together.

The written examinations occupy two days, and the oral and laboratory and outdoor examinations three to four days.

Candidates must send in their names and pay the fees a fortnight before the examination. Examination fee is five guineas. Re-examination fee one guinea.

FEES.

Arrangements have been made, in conjunction with the other Scottish Universities, for the institution of an inclusive fee for the courses of instruction leading to the M.B. and Ch.B. degrees. The inclusive fee for instruction within the walls of the University is ninety guineas, payable in five annual instalments.

The cost of matriculation, class and hospital fees for the whole curriculum, including the fees for the degrees, is usually about £160.

UNIVERSITY OF ST. ANDREWS.

The degrees conferred are Bachelor of Medicine and Bachelor of Surgery (M.B., Ch.B.), Doctor of Medicine (M.D.), and Master of Surgery (Ch.M.). The inclusive fee for the University instruction for M.B., Ch.B. is ninety guineas; and the inclusive fee for the clinical courses is forty guineas. These fees may be paid by annual instalments. For M.D. or Ch.M. the fee payable is fifteen guineas.

Two constituent colleges of the University provide medical teaching. The United College, St. Andrews, offers classes for two years, and the student may pass the first and second professional examinations at St. Andrews. There are excellent opportunities for combining degrees in Arts and Science with those of Medicine. Inclusive fees have been arranged for students who wish to take advantage of these opportunities. There are many bursaries offered to students who desire to graduate in Medicine, and it should be added that the cost of rooms and of living in St. Andrews is considerably less than in the larger University cities. For women there is an excellent residential hall, which is governed by the University authorities. The medical school is now carried on in buildings specially built for the purpose.

The Conjoint School of Medicine, Dundee, supplies a complete course of medical study, and the student from United College, St. Andrews, completes his curriculum there. Large new buildings with well-equipped laboratories have been provided. Both in the Medical School and the wards of the Dundee Royal Infirmary the students have unrivalled opportunities for gaining a practical knowledge of medical science and of medical work, for they have individual attention and supervision which the larger schools cannot give.

The Dundee Royal Infirmary contains 400 beds, and includes special wards for obstetrics, gynecology, children's diseases, ophthalmology, dermatology, otology, incipient insanity, and electrical therapeutics. New out-patient departments are now in use. There is a large out-door maternity department. Hospital Fees—Surgical and Medical, £3, 3s. yearly; Perpetual Ticket, £10, or in instalments, £10, 10s.; Obstetric Cases, £2, 2s.; Obstetric Clinic, £1, 1s.

Westgreen Asylum at Liff provides abundant material for instruction in

mental diseases, and the City Fever Hospital in fevers. The Dundee Eye Institution furnishes cases for instruction in ophthalmology.

The Diploma of Public Health (D.P.H.) may be taken at the Combined School of Medicine, Dundee.

A Diploma in Dental Surgery (L.D.S.) is also granted by the University. Instruction in the University, Royal Infirmary, and Dundee Dental Hospital.

All classes in the University are open to men and women alike.

UNITED COLLEGE, ST. ANDREWS.

PROFESSORS AND LECTURERS.

Physics— Professor Butler, M.A.
Chemistry— Professor Irvine, Ph.D., D.Sc.
Zoology— Professor M'Intosh, M.D., LL.D., F.R.S.
Botany— R. A. Robertson, M.A., B.Sc.
Physiology—Professor Herring, M.D.
Anatomy— Professor D. Waterston, M.D., F.R.C.S.

UNIVERSITY COLLEGE, DUNDEE.

PROFESSORS AND LECTURERS.

Physics— Professor Peddie, D.Sc.
Chemistry— Professor Mackenzie, D.Sc.
Zoology— Professor Thompson, M.A., D.Litt., C.B.
Botany— Professor Geddes, F.R.S.E.
Physiology—Professor Waymouth Reid, M.B., Sc.D., F.R.S.
Anatomy— Principal Mackay, M.D., LL.D.
Lieut.-Col. Lamont, M.B., I.M.S. (retired).
Surgery— Professor MacEwan, M.D., C.M.
Surgery, Clinical—
D. M. Greig, C.M., F.R.C.S.
L. Turton Price, Ch.B., F.R.C.S.
Medicine— Professor Stalker, M.D.
Medicine, Clinical—
Professor Stalker, M.D.; J. Mackie Whyte, M.D.
Materia Medica—
Professor C. R. Marshall, M.D.
Pathology— Professor Sutherland, M.B.
Midwifery and Gynæcology—
Professor Kynoch, M.B., F.R.C.P., F.R.C.S.
Midwifery and Gynæcology, Clinical—
Professor Kynoch, M.B., F.R.C.S.
R. C. Buist, M.D.
Forensic Medicine—
C. Templeman, M.D., D.Sc., M.O.H.
Ophthalmology—
Angus MacGillivray, M.D., D.Sc.
Diseases of Ear, Nose, and Throat—
R. P. Mathers, M.D.
Diseases of Children—
D. M. Greig, C.M., F.R.C.S.
J. S. Y. Rogers, M.B.

Diseases of Skin—
W. E. Foggie, M.D.
Mental Diseases—
W. Tuach Mackenzie, M.D.
Vaccination—
R. C. Buist, M.D.
Clinical Pathology—
F. M. Milne, M.B., D.P.H.
Clinical Medical Tutor—
Charles Kerr, M.B.
Clinical Surgical Tutor—
W. L. Robertson, Ch.B., F.R.C.S.
Anæsthetics—
A. Mills, M.D.
Bacteriology
W. J. Tulloch, M.D.
Dean of the Faculty of Medicine—
Professor Kynoch.

QUALIFICATIONS GIVEN BY THE SCOTTISH COLLEGES.

The Royal College of Physicians of Edinburgh, the Royal College of Surgeons of Edinburgh, and the Royal Faculty of Physicians and Surgeons of Glasgow, conjointly confer the Triple Qualification (L.R.C.P.E., L.R.C.S.E., L.R.F.P.S.G.). Female candidates are admitted to the examinations for this qualification.

Preliminary Examination.—This examination must be passed before the student commences professional study. It may be passed before any of the Boards recognised by the General Medical Council, and enumerated in the Regulations of the Colleges. The Educational Institute of Scotland conducts a qualifying Preliminary examination for medical students, in Edinburgh and Glasgow, on behalf of the Colleges. This examination embraces English, Latin, Mathematics, and either Greek, French, German, Italian, or other modern language. All the subjects may be passed at one or not more than two times. Calendar, containing examination papers, can be had from Mr. Hugh Cameron, M.A., 34 North Bridge Street, Edinburgh. Price 1s.

Professional Education. The curriculum must extend over five years. Graduates in Arts or Science of any recognised University who have spent a year in the study of Physics, Chemistry, and Biology, and have passed an examination in these subjects for the degrees in question, are exempted from the first year of study. The fifth year of study should be devoted to clinical work in one or more recognised Hospitals or Dispensaries, and to the study of special diseases. For information regarding the payment of class fees by the Carnegie Trust, *vide* p. i.

Order of Study which is Recommended.

First Summer— Physics and Elementary Biology.
First Winter— Five months' course in Chemistry and Anatomy; three months' course in Practical Chemistry; Practical Anatomy.
Second Summer— Practical Anatomy and Lectures; Practical Physiology.
Second Winter— Practical Anatomy; Physiology—Five months' course.
Third Summer— Three months' course in Practical Pathology, Materia Medica, and Practical Materia Medica; Surgical Hospital Practice.

Third Winter— Six months course in Surgery and Clinical Surgery; Attendance at Surgical Wards; Anæsthetics; Pathology.

Fourth Summer— Three months' course in Midwifery, in Gynæcology, in Medical Jurisprudence and Public Health, and in Clinical Surgery; Hospital Practice.

Fourth Winter— Six months course in Medicine and Clinical Medicine; Hospital Practice.

Fifth Summer— Three months at Clinical Medicine; Hospital; Insanity; Diseases of Children; Diseases of Eye.

Practical Midwifery—Personal attendance on twelve cases under the supervision of a medical practitioner, *or* three months' attendance at a Lying-in Hospital and personal attendance on six cases.

Fifth Winter— Hospital Practice; Fevers; Dispensary; Vaccination; Skin Diseases; Ear and Throat Diseases; Eye Diseases; Venereal Diseases; Operative Surgery.

Professional Examinations.—Four of these are held during the curriculum. Each is held quarterly in Edinburgh and twice a year in Glasgow. Candidates may enter for all or any of the subjects at the First, Second, and Third Examinations. In the Final Examination the subjects of Medicine, Surgery and Midwifery shall be taken together at the conclusion of five Winters and five Summers of Medical Study, provided that a period of twenty-four months has elapsed since passing the Second Professional Examination; and the subject of Medical Jurisprudence and Public Health may be taken at any time after passing the Third Examination. Candidates are advised to enter for the entire examinations.

First Examination— Physics, Chemistry and Elementary Biology. This should be passed before the beginning of the second winter session.

Second Examination— Anatomy, Physiology, including Histology. This should be passed at the end of the second year of study.

Third Examination— Pathology, Materia Medica and Pharmacy. This should be taken at the end of the third year.

Final Examination— Can only be taken at the end of the fifth year. The candidate must have attained the age of twenty-one. It includes—

1. Medicine, Therapeutics, Medical Anatomy, Clinical Medicine.
2. Surgery, Surgical Anatomy, Clinical Surgery, Diseases and Injuries of the Eye.
3. Midwifery and Diseases of Women.
4. Medical Jurisprudence and Public Health. This can be taken any time after the Third Examination.

Fees for Professional Examinations.

For each of the first three, £5; for the final, £15. The minimum total expense, inclusive of fees for classes and examinations, amounts to £115.

Fees for examinations *in Edinburgh* should be lodged with Mr. D. L. Eadie, 50 George Square, and *in Glasgow* with Mr. Walter Hurst, 242 St. Vincent Street.

Diploma in Public Health of the Royal Colleges.

The Diploma is granted by the Triple Qualification Board.

1. Every candidate for examination must hold a registrable medical qualification, which has been registered under the Medical Acts

2. After obtaining such qualification he must have attended a recognised Laboratory in which Chemistry, Bacteriology, and the Pathology of the Diseases of Animals Transmissible to Man are taught; and the certificate must show that the candidate has conducted Chemical and Bacteriological analyses of air, water, sewage and foods, and certify that the candidate has attended not less than four calendar months, and that he has worked in the Laboratory for at least 240 hours, of which not more than one-half shall be devoted to Practical Chemistry.

3. After obtaining a registrable qualification he must during six months (of which at least three months shall be distinct and separate from period of Laboratory instruction required) have been engaged in acquiring a practical knowledge of the duties of Public Health Administration for not less than sixty working days under the personal supervision of—

(*a*) In England or Wales, the Medical Officer of Health of a County or single sanitary District having a population of not less than 50,000, or a Medical Officer of Health devoting his whole time to Public Health work; or

(*b*) In Scotland or Ireland, the Medical Officer of Health of a County or District or Districts with a population of not less than 30,000; or

(*c*) In Ireland, a Medical Superintendent Officer of Health of a District or Districts having a population of not less than 30,000; or

(*d*) In the British Dominions outside the United Kingdom, a Medical Officer of Health of a Sanitary District having a population of not less than 30,000, who himself holds a Registrable Diploma in Public Health; or

(*e*) A Medical Officer of Health who is also a Teacher in the Department of Public Health in a recognised Medical School.

(*f*) A Sanitary Staff Officer of the Royal Army Medical Corps having charge of an Army Corps, District, Command, or Division recognised for the purpose by the General Medical Council.

4. After obtaining a medical qualification he must have attended for three months at least twice weekly the practice of a Hospital for Infectious Diseases, at which he has received instruction in the methods of administration.

The examination consists of two parts. The first part includes (*a*) Laboratory work, with Chemistry and Bacteriology; (*b*) Physics and Meteorology.

The Second Examination embraces—(*a*) Report on premises visited; (*b*) Examination at Fever Hospital; (*c*) Examination at Public Abattoir; (*d*) Epidemiology and Endemiology; (*e*) Vital Statistics and Sanitary Law; (*f*) Practical Sanitation.

Each examination is held bi-annually, in October and May. The fee for each is £6, 6s.; for re-examination, £3, 3s. Fees and applications to be lodged with Mr. D. L. Earlie, 50 George Square, Edinburgh; or with Mr. Walter Hurst, 242 St. Vincent Street, Glasgow.

Membership and Fellowship of the Royal College of Physicians, Edinburgh.

Every applicant for the *Membership* must possess a recognised qualification, and be not less than twenty-four years of age. He must pass an examination on Medicine and Therapeutics, on Clinical Medicine, and on some Special Department of Medicine, such as Psychological Medicine, General Pathology and Morbid Anatomy, Medical Jurisprudence, Public Health, Midwifery, Diseases of Women, Diseases of Children, Tropical Medicine, etc. The Membership is conferred by election.

The fee for the Membership is thirty-five guineas, except the applicant be a Licentiate of the College, when it is twenty guineas.

Members of not less than three years standing may be raised by election to the *Fellowship*, the fee being thirty-eight guineas, exclusive of Stamp Duty of £25.

FELLOWSHIP OF THE ROYAL COLLEGE OF SURGEONS, EDINBURGH.

Every candidate must be twenty-five years of age, and must have been engaged for two years in the practice of his profession, after having obtained a recognised qualification in Surgery. The petition for examination must be signed by two Fellows—a proposer and seconder.

The candidate must pass an examination on Principles and Practice of Surgery, including Surgical Anatomy, Clinical Surgery, and any one of the optional subjects: Ophthalmic Surgery, Aural, Nasal and Laryngeal Surgery, Dental Surgery, Surgical Pathology and Operative Surgery, Gynæcology, Advanced Midwifery with Obstetric Surgery, Advanced Anatomy.

The fee is £45, except the candidate be a Licentiate of the College, when the fee is £35. Further particulars may be obtained from the Clerk to the College, 50 George Square, Edinburgh.

FELLOWSHIP OF THE ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW.

Every candidate must have been qualified for two years, and be aged twenty-four. Admission to the Fellowship is by examination and subsequent election. The candidate is examined on either (*a*) Medicine (including Clinical Medicine, Medical Pathology, and Therapeutics), or (*b*) Surgery (including Clinical Surgery, Operative Surgery, Surgical Anatomy, and Surgical Pathology); and on one optional subject—Anatomy, Physiology, Pathology, Midwifery, Diseases of Women, Medical Jurisprudence, Ophthalmic Surgery, Aural, Laryngeal and Nasal Surgery, Dental Surgery, State Medicine, Psychological Medicine or Dermatology.

The fee is £30, except the candidate be a Licentiate of the Faculty, when it is £15.

The Fellowship Diploma is now open to Women.

POST-GRADUATION STUDY.

The different University Laboratories provide facilities for research work. In most cases no fees are charged, but those engaged in research work are expected to defray the expense of materials. The laboratory of the Royal College of Physicians of Edinburgh is splendidly equipped for the carrying out of all branches of medical research. It is available for research work to Members and Fellows of the Royal Colleges of Edinburgh and to other applicants approved by the Council of the Royal College of Physicians. No fees are charged, and the ordinary reagents, etc., are provided. By arrangement with the Superintendent, workers may have the assistance of members of the Laboratory Staff. Special post-graduate courses may be arranged.

EDINBURGH POST-GRADUATE COURSES IN MEDICINE

These courses, which are held during the Summer Vacation, are arranged by a Committee appointed by the University and School of the Royal Colleges. The programme for 1917 has not yet been drawn up, but will no doubt be somewhat similar to that for 1914, which was as follows:—

JULY.—A series of classes dealing with *Diseases of the Nervous System* (fee, Seven Guineas), and another series on *Diseases of Children* (fee, Five Guineas), were arranged for during the last fortnight of the month.

AUGUST.—A Four Weeks' Course on *Internal Medicine*. This included two clinics daily upon diseases of the various systems, in addition to classes upon Applied Anatomy, Hæmatology, Bacteriology and the Examination of the Heart, Urine, Digestive Products and Nervous System, X-Ray Diagnosis, and the Medical Aspects of Morbid Pregnancy. Between six and seven hours' instruction daily. Attendance limited to twenty-five. *Fee*, Ten Guineas.

SEPTEMBER:—A Four Weeks' *General Course*. Each fortnight was quite independent and might be taken separately. This included Medical and Surgical Clinics, Clinical Neurology, Dermatology, Fevers, Ophthalmology, Pediatrics, Infant Feeding, Applied Anatomy, Morbid Anatomy, Pathological Histology, etc. *Fee*, Five Guineas for the month or Three Guineas for either fortnight. A series of lectures upon subjects of general interest was given in connection with the course. These lectures were open to all practitioners.

A Four Weeks' *Surgical Course* included Surgical Clinics, Applied Anatomy, Surgical Pathology, Operative Surgery, etc. Attendance limited to thirty. *Fee*, Ten Guineas.

A Special Surgical Course during the second fortnight of September on the *Genito-Urinary Tract*. *Fee*, Six Guineas.

A Course on the *Ear, Nose, and Throat* (limited to ten) included classes on the pathology of this subject and operative work in addition to demonstrations on the methods of examination, clinics, etc. *Fee*, Ten Guineas.

A *Series of Classes*, the entries for which were limited, upon Hæmatology, Bacteriology, X-Rays, Gynecology, Gynecological Pathology, Ophthalmoscopy, Errors of Refraction, Ear, Nose and Throat, Histological Methods, etc. These classes were open only to those who had entered for the General, Surgical, or Ear, Nose and Throat courses, on payment of an Additional Guinea in each instance.

The various courses are attended by numbers of men which every year for some summers past have shown a steady increase. To ensure places for the limited courses it is necessary in some cases to enter at least three months before the beginning of the course desired.

In addition to these courses the classes throughout the year on Bacteriology, Diseases of the Blood, Diseases of the Tropics, Neurology, etc., are attended by a number of graduates.

Particulars of these classes are to be had from the Secretary, The New University.

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Edinburgh medical journal

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